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FOR THE STUDY OF CARDIOLOGY AND THE CIRCULATION

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CHARLES LAUBRY

It is with great regret that we learn, from a brief notice in a Swiss journal, of the death at Nantes of Professor Charles Laubry, of Paris.

The great traditions of the Paris school of cardiology have been handed down in direct line from master to pupil for over a century, starting with Corvisart and passing in turn to Bouillaud, to Potain, to Vaquez, and finally to Laubry. All these have been masters of bedside teaching and clinical observation. In our own time, the physicians trained and inspired by Vaquez, among whom Laubry was prominent, have exercised a powerful influence on cardiological teaching, especially in Latin Europe and Latin America, comparable in some ways with Mackenzie's influence in English-speaking countries. In 1908, Vaquez, assisted by Laubry and Aubertin, founded the *Archives des Maladies du Cœur*, which for over thirty years has been a mainstay of French cardiology.

Graduating from the école Vaquez, Laubry established his own clinic, which, after several moves, eventually settled at the Hôpital Broussais in 1925. In this ancient and dilapidated building, situated in a poor quarter of the south of Paris, and almost unaltered since the war of 1870, Laubry organized a complete cardiological centre. Here it was my privilege to pass a month or so in 1926, when Laubry's reputation as a teacher was beginning to attract visitors from abroad.

Laubry was a man of most attractive personality. Overflowing with good nature, paternal in manner, animated in speech, he possessed in full measure all those qualities that we most admire as typically French. He was, above all, a great clinical teacher, at his best at the bedside or in the out-patient department. Here he would give impromptu a polished and complete clinical lesson without ever becoming tedious, so that one finished a ward round with the feeling that time had passed all too quickly.

Though by no means slavishly accepting all the views emanating from the Vaquez clinic at the Pitié, and rejecting, for example, the elaborate mensuration of the orthodiagram practised by Bordet, Laubry was unwavering in his loyalty and devotion to his old chief, to whom he delighted to pay public tribute. His interests covered the whole field of cardiovascular problems, and it is difficult to single out any particular aspect of his work; perhaps his contributions to the radiology of the heart deserve special mention.

Early convinced of the importance of X-ray examination through his association with Vaquez and Bordet, Laubry was fortunate in having as his assistant Robert Chaperon, a pioneer worker in this particular field; it was he

who developed the technique of radio-opaque injection of the individual heart cavities and great vessels *in situ* in the cadaver, as a method of studying the anatomy of the heart; and important papers on the vascular pedicle and the composition of the lung root shadows resulted. After the untimely death of Chaperon, this work was continued and extended in co-operation with Heim de Balzac, and the fruits of many years of patient investigation were incorporated in the large treatise "*Radiologie Clinique du Cœur et des Gros Vaisseaux*," published by Laubry and his associates in 1939 shortly before the war. This book will certainly have a permanent value in reference to all aspects of the radiological anatomy of the heart, normal and pathological.

Though his reputation rested more on the spoken than on the written word, Laubry wrote much. His book "*Traité des Maladies Congénitales du Cœur*," written jointly with Pezzi, appeared in 1921 and is still widely quoted. His "*Leçons de Sémiologie Cardiovasculaire*," published in 1924, is perhaps his best-known book, and makes delightful reading, bearing comparison with the famous clinical lessons of Merklen. In 1930, he published a large text-book on heart disease, which, though excellently produced, was rather a bulky volume and never attained the popularity of Vaquez' text-book.

No account of Laubry's work would be complete without some mention of the "*bruit de galop*." This sign, long neglected in England, had been described in masterly fashion by Potain in 1875, and seemed to possess a singular fascination for Laubry, who paid great attention to it in his teaching. With Pezzi, he wrote a monograph on gallop rhythm in 1926, and also made it the subject of his St. Cyres Lecture, delivered in London in 1937.

Laubry was never a *professeur agrégé*, and was not therefore normally eligible for a professorial chair; but when the time drew near for him to retire as a *Médecin des Hôpiteaux*, a special chair of Clinical Cardiology was created so that his services as a teacher could be retained. He accomplished much for French cardiology besides his purely scientific contributions. After the death of Vaquez, he took over the direction of the *Archives des Maladies du Cœur*, and greatly improved the standard of its production, at the same time excluding from its scope diseases of the blood. In 1937 he initiated the Société Française de Cardiologie, of which he was nominated first president, Prof. Clerc and Dr. Gallavardin being joint vice-presidents. Whereas the *British Heart Journal* was started by the Cardiac Society, in France the reverse occurred, for the society was formed by the journal through its editorial committee. Laubry next envisaged the formation of an international society of cardiology, and broached the subject on the occasion of a dinner given in his honour in London in 1937. Unhappily events were to prove unfavourable to his project.

All who knew Laubry will wish to join with me in paying affectionate tribute to his memory, and all members of the Cardiac Society will share our regret in the passing of another of our distinguished honorary members.

D. EVAN BEDFORD.

TRICUSPID STENOSIS;
WITH PARTICULAR REFERENCE TO DIAGNOSIS AND PROGNOSIS

BY

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Recent experience with several cases of tricuspid valve disease, attending out-patient clinics without arousing any thought of this valve lesion, has stimulated our interest in the subject and has emphasized the inadequacy of most discussions on the subject.

It has become almost traditional to state that tricuspid stenosis is rare and that the diagnosis is difficult. Herrick (1897) wrote that the disease is so rare that the full details of every case should be reported. Mackenzie (1908) wrote that he had heard a tricuspid murmur only three times in his life. Osler and Gibson (1915) advocated caution in the diagnosis—" As a rule the physician is in a safer position if he limits his diagnosis to two valves: clinically, when lesions of three or four valves are determined with accuracy, mortifying post-mortem disclosures are not unlikely to follow." Lewis (1933) rather implies that the diagnosis is not worth making when he writes " I have not known the diagnosis, when made, affect the management of any case." Strümpell and Seyfarth (1928) say that tricuspid valve disease is so rare as to have no practical importance. The stress that has been laid on the rarity and difficulty of diagnosis of the affection combined with statements such as those above, have tended to make the average practitioner forget that the tricuspid valve is not infrequently affected. Of the 250 reported cases collected by Zeisler (1932) the diagnosis had been made before death in only 31: of those, 14 were made by Dressler and Fischer (1929–1930) who reported 33 autopsied cases, but in their series it was only after their interest had been aroused that the diagnosis began to be made, resulting in the correct interpretation in 11 out of the last 14 cases.

We have, therefore, thought it worth while to consider if the diagnosis of tricuspid stenosis is after all entirely unimportant and to determine as clearly as possible the diagnostic criteria.

According to the pathologist, the tricuspid valve becomes stenosed when the ostium is reduced to between 10 and 11 cm., the normal being between 12 and 14 cm. (Cabot, 1926). White (1937) writes that stenosis of clinical importance

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and of such degree as to be diagnosable clinically in some cases is not reached until the ostium is reduced to 8 cm. or less.

The valve becomes incompetent for three reasons: (1) extensive scarring in which regurgitation accompanies stenosis; (2) slight shortening of the chordæ tendineæ or fibrosis of the valve edge resulting in regurgitation, without necessarily causing stenosis; and (3) dilatation of the valve ring due to myocardial infection, long standing congestive failure, or constitutional disease such as anæmia. The valve is diseased in the first and second instances, and defective though not diseased in the third; the functional disorder of the third instance may actually be more serious than organic valvular disease, at least as a manifestation of important trouble, though often it is only a transient condition.

Incidence and Pathology

A false idea of the incidence of tricuspid valve stenosis has been given by the numerous compilations of isolated case reports of the disease. There exists, moreover, a good deal of discrepancy between several of the analyses that have been published, doubtless dependent largely on the degree of stenosis. In Baltimore only 7 cases were found in 24,000 autopsies (Hirschfelder, 1910). Coombs (1924) gives the following figures of the incidence of valve injury in 97 cases of rheumatic heart disease, including the lesser as well as the greater degrees of involvement.

Total	Mitral	Aortic	Tricuspid	Pulmonary
97	97	57	35	2

These were combined in the following ways:

Mitral alone	27 cases
Mitral and aortic	35 ,,
Mitral and tricuspid	12 ,,
Mitral, aortic, and tricuspid	21 ,,
Mitral, aortic, tricuspid, and pulmonary	2 ,,

He does not give the relationship of these cases to the clinical signs, but later states that the incidence at Bristol of clinical tricuspid stenosis in rheumatic heart disease was 14 per cent. Cabot (1926) gives the incidence at 15 per cent while Dressler and Fischer put the incidence at 24 per cent.

Bland, Jones, and White (1935) analyzed the pathological findings in 100 cases of fatal rheumatic disease below the age of 21 in whom the diagnosis of tricuspid valve disease had not been made during life. Out of 100 cases, the mitral valve was affected in 98 instances, the aortic in 71, the tricuspid in 30, and the pulmonary valve in 5, and there was one case with no valves at all affected. The valve lesions, many of them acute with little or no deformity, occurred alone or combined as follows:

Mitral alone	23 cases
Aortic alone	1 case
Mitral and aortic	45 cases
Mitral and tricuspid	5 ,,
Mitral, aortic, and tricuspid	20 ,,
Mitral, aortic, tricuspid, and pulmonary	5 ,,

This group is selected, in that it is composed of cases dying of rheumatic fever: therefore one might suggest that they had more extensive heart involvement than a similar group having rheumatism over the same period of time, but surviving; and that therefore the incidence may naturally be expected to be higher. On the other hand, valve deformity sufficient to produce stenosis is less common in these younger cases, even though fatal; the myocardium is more involved than the valves. Yet it is fairly certain that cases surviving show a rather high incidence of tricuspid involvement, even though rarely progressing to a degree of much stenosis. Von Glahn (1927) examined 109 rheumatic heart cases at autopsy, grouping them into acute or chronic lesions, and found 19 acute and 26 chronic tricuspid valve lesions making a total of 45 or 41 per cent. Libman (1923) has always emphasized the high incidence of tricuspid involvement (not stenosis as such) in rheumatic heart disease; in one small series, he found the valve involved in 12 out of 18 cases of endocarditis. Thayer (1925) also found a high involvement (44 per cent). Cabot (1926) records 33 cases of tricuspid stenosis among 4000 autopsies between 1896 and 1919 at the Massachusetts General Hospital.

Our Series of Cases

We have analyzed the data on 30 cases of tricuspid stenosis proved post-mortem, and on 12 cases that we ourselves have studied during life—3 confirmed by autopsy, the others still alive but almost certainly correctly diagnosed. At the Massachusetts General Hospital between the years 1920 and 1937 inclusive, there have been 4300 autopsies; these included 217 cases of rheumatic heart disease, and in that series there was involvement of the tricuspid valve in 47 cases. Of these 47 there were 17 in whom the affection was probably of no clinical significance, being either an acute terminal rheumatic process or a bacterial endocarditis or one of the group that Libman states is common, slight scarring and fibrosis of a limited segment of the valve ring.

The valves affected in this whole group from 1920 to 1937 at the Massachusetts General Hospital consisting of 217 cases of rheumatic heart disease were as follows:

Mitral alone	59 cases
Aortic alone	11 ,,
Mitral and aortic	100 ,,
Mitral and tricuspid	7 ,,
Mitral, aortic, and tricuspid	35 ,,
Mitral, aortic, tricuspid, and pulmonary	5 ,,
Total	217 ,,

Thus there were 47 cases of tricuspid disease (22 per cent) among the 217, but only 30 (14 per cent) that were important in themselves (as definite tricuspid stenosis).

There is unfortunately no satisfactory way in the series of cases noted above of correlating the post-mortem data with the clinical signs of tricuspid disease that may have been present. The diagnosis was made before death in only 1 of the 30 cases and suspected in only 2 other cases. These 30 cases have

therefore been divided up by us quite arbitrarily into three clinical diagnostic groups according to whether or not the cases might have shown signs during life. Accordingly the clinical diagnosis justified by the data of the 30 cases of tricuspid stenosis was as follows:

1. Tricuspid stenosis and regurgitation of varying degree .. 19 cases
2. Possible functional impairment of tricuspid valve .. 9 ,,
3. Tricuspid ring dilatation alone .. .. 2 ,,

If these assumptions are correct, there would be roughly 9 per cent (that is, 19 out of 217 cases of rheumatic heart disease) of all cases of rheumatic valvular disease showing clinical signs of tricuspid stenosis and regurgitation.

There is one further fact to be considered statistically. In adults, tricuspid stenosis produces in its later stages a life of chronic invalidism, which lasts for very much longer than the invalidism produced by mitral valve disease alone. For this reason it is probable that these cases find their way to homes for the chronic sick rather than to the general hospitals, and so lower the post-mortem incidence of tricuspid disease in the hospitals.

As yet there is no large series of rheumatic heart cases that has been followed through from beginning to end, to give us a better idea of the true incidence. From the facts at present available, it is probable that one case in every ten of rheumatic heart disease of more than a few years' duration has organic disease of the tricuspid valve, at least moderate in degree.

Age and Sex

Herrick (1908) gives a summary of the ages at death of 187 reported cases of tricuspid stenosis: in his series and in our own series of 30 cases studied post-mortem they were as follows:

	Herrick's Series	Our Series
0–10 years	0	1
10–20 ,, ...	16	8
20–30 ,, ...	59	7
30–40 ,, ...	38	6
40–50 ,, ...	28	5
50–60 ,, ...	10	3
60–70 ,, ...	6	—
Age not given	30	—

Usually females have been more frequently affected than males (Duroziez, 1868; Herrick, J. B., 1897; Dressler and Fischer, 1929). In our 30 cases, 16 were males and 14 were females. In an additional later group of 12 cases that we have observed clinically, 7 were females and 5 were males.

Clinical Diagnosis

Duroziez (1868) reported 10 cases of tricuspid stenosis with ages varying from 22 to 64, and asserted that tricuspid disease was more common than was usually accepted. He was impressed by their marked cyanosis and by the fact that many could lie flat in bed in spite of gross cardiac deficiencies: in spite of marked valvular damage some were able to live to old age, and so he concluded

that the tricuspid lesion did not aggravate the mitral lesion. He drew attention to the systolic or diastolic murmur at the lower end of the sternum that pointed to the diagnosis, and wrote: "The disease should be diagnosed when the patient is a female, has a history of rheumatism, and of dyspnœa, palpitation, œdema, often with remissions and exacerbations, is cyanosed, has mitral stenosis with an enlarged right heart, particularly if the auricular enlargement can be made out. Especial value attaches to the persistent cyanosis. If in addition to this the patient has a separate murmur best heard over the ensiform or the fifth or sixth right costal cartilage, then the diagnosis becomes reasonably certain." This admirably summarizes the chief points. There is no one infallible diagnostic sign but there are numerous signs, any one of which should initiate a search for other clues.

The diagnosis may be suspected from the *history* alone as Levine and Thompson (1937) have pointed out. Any adult patient who has repeated attacks of œdema and ascites and yet is able to lead a sedentary life with the aid of diuretics and carry on with symptoms that ordinarily would cause the early demise of the patient, probably has tricuspid stenosis.

Several observers, most recently Wearn (1936), have commented on the peculiar colour, a mixture of jaundice and cyanosis, that is presented by these patients, pointing out that this should always suggest the possible diagnosis of tricuspid stenosis. Much reliance cannot, however, be placed on this sign inasmuch as its commonest pathogenesis lies in the inability of an engorged or diseased liver to excrete rapidly enough the blood pigment from a pulmonary infarct (so common in mitral stenosis with congestive failure), whether mitral stenosis alone or mitral stenosis with tricuspid valve disease is responsible for the trouble with the liver.

A patient with a rheumatic heart and ascites who is *able to sleep without extra pillows* probably has tricuspid stenosis. This is due to the lack of pulmonary vascular engorgement, as confirmed by X-ray study, resulting from the obstruction to the free flow of blood through the right heart.

Pulsation in the neck veins has for over one hundred years excited comment, with much argument as to whether the jugular polygram is diagnostic. Mackenzie (1894) pointed out that the deep (internal) jugular pulse in the neck is frequently thought to be arterial in origin because it is systolic in time. As a matter of fact it can very easily be shown to be venous in origin by light compression over the base of the neck (jugular bulb) with obliteration of the pulse (Fig. 1). The significance of this venous pulsation has recently been studied by us (White and Cooke, 1939). We found that although this pulse in the neck signifies only tricuspid regurgitation, whether due to organic disease or to functional regurgitation, *there is, nevertheless, a very strong probability, in fact almost a certainty, of the presence of tricuspid stenosis in the case of a patient in the fourth or fifth decade with rheumatic heart disease who has for years marked systolic deep jugular pulsation with little or no congestive failure* (see Cases 1 and 2, and Fig. 2).

The same conclusion holds good about *liver pulsation*. It is interesting that the obse rvation is frequent in the follow-up records of a large number of the

histories we have studied, " that the patient still shows some signs of failure as evidenced by his large liver." On the other hand tricuspid stenosis may be

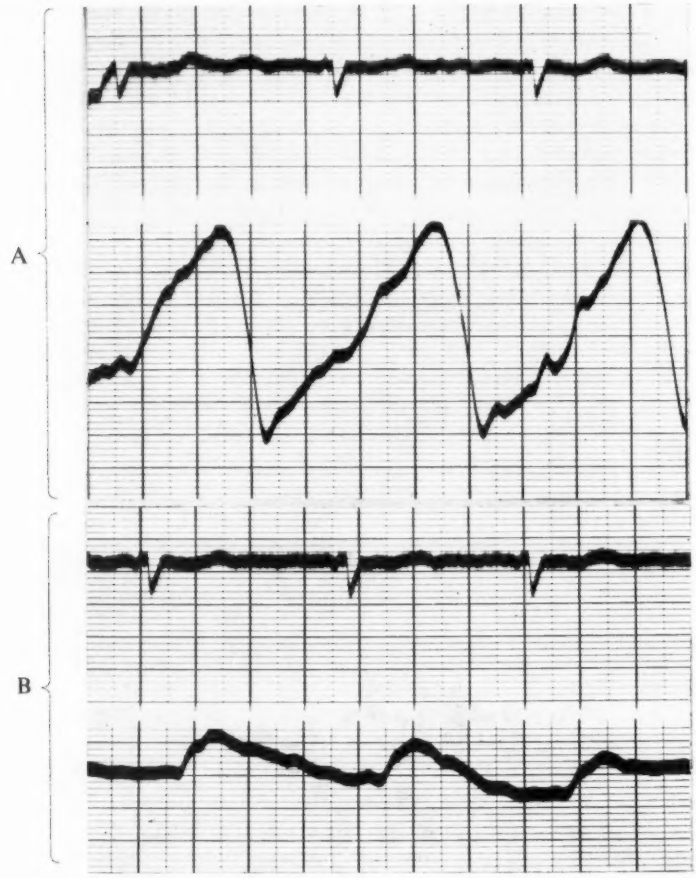


FIG. 1.—(A) Simultaneous tracings of electrocardiogram (lead 1), above, and of deep jugular pulsation, below, in a case of rheumatic, aortic, mitral, and tricuspid stenosis, with auricular fibrillation. Note the slow upward stasis curve with superimposed systolic pulse.

(B) Simultaneous tracings of electrocardiogram (lead 1), above, and of deep neck pulsation, below, after compression of the jugular bulb. The receiving cup has not been moved in its position. The jugular pulse has been obliterated and only the underlying carotid pulse remains (it is anacrotic).

present without venous pulsation either in the neck or in the liver (Clements, 1927; and others).

Tschilikin (1930) states that the only pathognomonic sign of tricuspid stenosis is a *localized diastolic murmur* at the lower end or to the right of the sternum. However, this sign is often, probably usually, absent, as in the

FIG. 2.—Case 3. (A), (B), (C) Sequential exposures taken from a moving picture film of the systolic deep jugular pulse of a young man with tricuspid stenosis: recorded in the sitting position. Early diastole in A, full systole in C, and midway in B.

majority of Dressler and Fischer's cases. It was present in 7 of the 12 cases that we have ourselves examined clinically.*

Auricular fibrillation and polycythaemia occur just as frequently in cases of pure mitral valve disease, especially the so-called " tight type," as in tricuspid stenosis. There is no evidence in the group of cases here reported that pulmonary infarcts are more frequent with tricuspid stenosis than in mitral stenosis alone.

No diagnostic significance can be attached to the electrocardiogram.

We have estimated the *venous blood pressures* and *circulation rates* in seven of these patients. All the cases, although ambulatory and comparatively well, gave readings between 19 and 27 cm. of water (method of Burwell *et al.*). This has been noted in the absence of œdema, constrictive pericarditis, and mediastinal obstruction on several occasions (e.g. Friedlander and Kerr, 1936; Altschule and Blumgart, 1937) and should be taken as a strong diagnostic point in favour of tricuspid stenosis. The circulation times were all markedly prolonged, the right heart times averaging 20 seconds (ether method) against a normal average of 6 seconds.

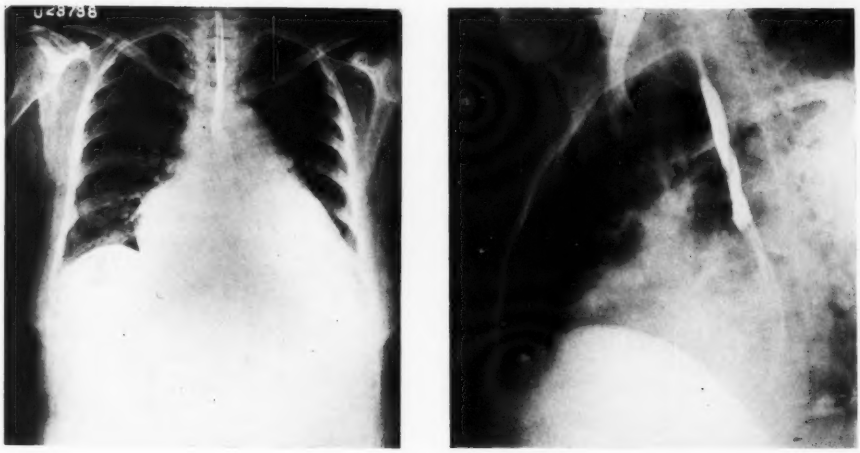
Dressler and Fischer emphasized as an important diagnostic pointer the presence of *marked enlargement of the heart to the right by X-ray* especially when associated with absence of pulmonary congestion at the hilus of the lung.

Lian and Marchel (1936) drew attention to the *deviation of the œsophagus to the left* in the presence of marked enlargement of the heart to the right. They suggested that this finding, while not pathognomonic of tricuspid stenosis, indicates enlargement of the right auricle and should lead to the suspicion of tricuspid stenosis. We have found in 4 of the cases here reported that the œsophagus is deviated to the left and in other instances appears to travel down the front of the spinal column in the midline. Certainly the incidence of this left deviation of the œsophagus in our small group of cases warrants some consideration being paid to this finding (Fig. 3).

Castex, Battro, and Quirno (1939) reported two cases of tricuspid disease diagnosed during life. Kymograms of both patients suggested the diagnosis of tricuspid regurgitation by the presence of systolic pulsation in the right auricle and in the dilated superior vena cava.

We are herewith presenting the histories and essential findings in 17 of our cases, the first 3 in some detail as classical examples of the condition and the last 14 in brief. Details are summarized in the table on p. 157. In the 217 autopsies of patients with rheumatic hearts already referred to, there were 5

* An interesting possible but doubtless very rare explanation for a middiastolic murmur at the lower end of the sternum, not transmitted from the apex and not due to organic tricuspid stenosis, is a mechanism in the right ventricle similar to that in the left ventricle which causes the Austin Flint murmur, namely, pulmonary regurgitation with dilated right ventricular cavity. It happens that recently (December 1939) one of us (P. D. W.) has encountered such a case, a patient in failure with marked mitral stenosis, functional pulmonary regurgitation giving rise to a Graham Steell murmur, and a well marked localized middiastolic rumble in the 4th intercostal space at the left border of the sternum just below and distinctly different and separate from the pulmonary systolic murmur. Tricuspid stenosis was diagnosed antemortem on this basis but not found at autopsy, which disclosed marked mitral stenosis, dilated partially thrombosed pulmonary artery and left auricle, and dilated right ventricle.



A B

FIG. 3.—(A) Antero-posterior X-ray view of the heart shadow of case of mitral and tricuspid stenosis showing barium-filled œsophagus deviated to the left.
(B) Left (II) oblique view of same case.

who had marked or clinically " pure " tricuspid stenosis, and 25 others with slight to moderate tricuspid stenosis, making a total of 30 cases.

CASE REPORTS

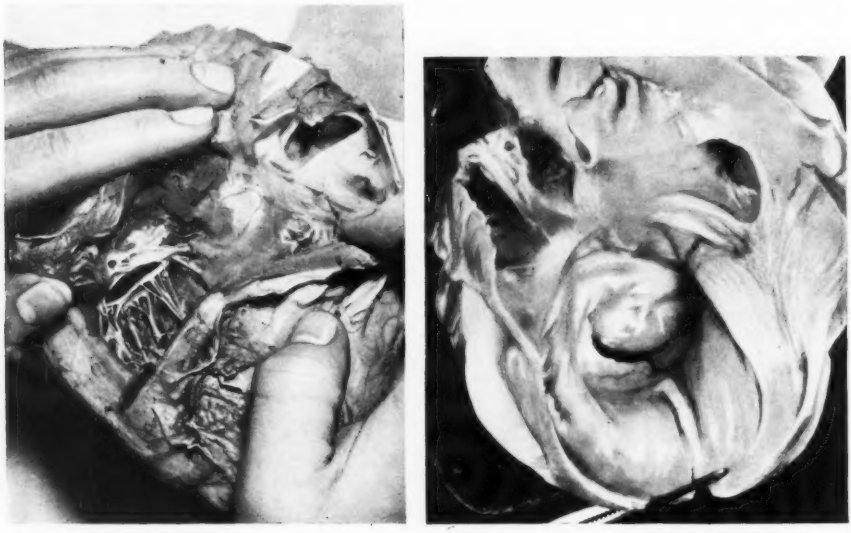
Case 1, female, aged 54 years. An attack of rheumatism at the age of 32 kept her in bed a short time, but she was back at work within three weeks. She first attended Massachusetts General Hospital in December 1935, for abdominal swelling of 18 months' duration. Mitral, tricuspid, and aortic stenosis and regurgitation were diagnosed. Since then she had carried on her housework and had entered the hospital every two or three weeks for abdominal paracentesis and occasional doses of salyrgan intravenously. Her condition remained fairly constant for the three years that she was under observation. She never used more than one pillow at night although the veins of the neck, forehead, and arms were remarkably distended when she lay down. She was slightly cyanosed and slightly icteric. The neck veins showed a systolic deep jugular pulsation extending to the angle of the jaw and easily obliterated by light pressure over the jugular bulb at the base of the neck. There was no engorgement of the superficial veins. Venous pressure was 26 cm. water. Circulation time: ether, 20 seconds (normal 5–10); and saccharin, 50 seconds (normal 15–25).

The heart was enlarged both to right and left and the œsophagus was deviated in its lower half to the left of the vertebral column. B.P., 115/85 mm. On auscultation there were systolic and mid-diastolic murmurs at the apex, and a loud aortic systolic murmur with a palpable thrill at the base, but no tricuspid murmur could be defined. E.C., auricular fibrillation with moderate right axis deviation, slight slurring of the QRS waves, and " digitalis " T waves.

She was admitted to the hospital for the last time after a cerebral vascular accident and died a few days later in April 1939.

Autopsy.—The superior and inferior vena cavæ were much dilated, measuring 2·8 and 4 cm. respectively in diameter. The heart weighed 410 g. The right auricle

was much dilated and its walls hypertrophied. The cardiac apex was formed by the large right ventricle although the walls were not thicker than those of a normal right ventricle. The tricuspid valve admitted two fingers only and measured 10·5 cm. in circumference (Fig. 4A); all the leaflets were thickened and fused to form a slightly



A B

FIG. 4.—Case 1. (A) Markedly stenosed (button-hole) tricuspid valve seen from below, in a case of chronic rheumatic heart disease.
(B) Stenosed mitral valve seen from above, in the same case.

nodular band 12–13 mm. in width; the chordæ were not appreciably thickened. The left auricle was also much dilated, approximating in size that of the right. The mitral valve was of the fish mouth type, 6 cm. in circumference but with remarkably narrowed and rigid fused cusps and an opening approximately 2·5 by 0·5 cm. (Fig. 4B). The aortic valve was approximately 7 cm. in circumference, with fusion of the valve cusps and poor approximation. The liver was enlarged and showed cirrhosis which was thought to be cardiac in origin.

Case 2, female, aged 49. No history of joint pains, rheumatic fever, chorea, or scarlet fever during childhood. At the age of 14, she had a chest cold which confined her to bed for a fortnight and left her very weak for some months. She was, however, able to play games actively as a child. At the age of 22 she began to notice some undue shortness of breath while doing heavy housework, and began to complain of attacks of praecordial pain which have been present more or less ever since. At the age of 27, she was rejected for life insurance owing to a murmur in her heart, of which she had been unaware. At 43 years she had some signs of congestive failure which cleared up but have recurred from time to time ever since. The following year she was admitted to the hospital with slight congestive signs and menorrhagia due to fibroids. She was digitalized and was able to continue her work in the household and at a sewing centre. A diagnosis of tricuspid stenosis was queried. At the age of 47, she was again admitted with abdominal swelling of two years' duration and examination at that time showed cyanotic lips, malar flush, distension of the neck veins, markedly engorged

TABLE SHOWING IMPORTANT FINDINGS IN 17 CASES OF TRICUSPID STENOSIS

	*Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8	Case 9	*Case 10	Case 11	Case 12	*Case 13	*Case 14	*Case 15	*Case 16	*Case 17
Age	54	49	40	54	60	36	57	50	39	34	58	29	21	41	42	30	41
Sex	F.	F.	M.	M.	F.	F.	M.	M.	F.	F.	M.	F.	M.	F.	F.	F.	F.
Rheumatic fever	+	0	0	+	+	+	0	+	+	+	+	+	+	0	?	0	0
Chorea	0	0	0	0	0	0	0	0	0	0	0	+	0	+	0	0	+
Duration of symptoms (in years)	5½	6	8	?	10	6	3	2	5	1½	4	?	½	7	?	2½	5
Cyanosis	sl.	sl.	+	sl.	sl.	sl.	0	0	sl.	sl.	0	sl.	+	+	+	sl.	+
Jaundice	sl.	0	0	0	sl.	sl.	0	0	sl.	sl.	0	0	0	0	0	0	0
Systolic deep jugular pulsation	++	+	++	+	+	+	+	+	+	+	+	+	+	—	—	0	+
Enlargement of liver	++	+	+	+	+	+	+	+	+	+	+	+	+	0	+	+	+
Pulsation of liver	+	+	+	+	+	+	+	+	+	+	0	+	+	0	+	+	+
Ascites	++	0	++	0	+	+	+	+	+	+	+	+	+	0	+	+	0
Diastolic murmur localized at lower end of sternum	0	+	+	+	0	0	+	0	0	+	+	+	0	0	0	—	+
Enlarged right heart	+	+	+	+	+	+	+	+	+	—	—	+	—	+	+	—	+
Deviation of œsophagus to left of midsternum (X-ray)	+	mid line.	+	—	—	+	+	—	mid line.	—	—	—	—	—	—	—	—
Arterial pressure (mm. mercury) systolic	115	150	100	150	235	120	130	120	110	135	120	120	140	115	150	120	125
diastolic	85	80	70	80	100	80	80	90	70	80	90	70	55	80	80	75	85
Venous pressure (cm. water)	26	22	24	22	21	—	—	19	24	—	—	—	—	—	—	—	—
Circulation time (sec.); saccharin (arm to tongue)	50	30	—	45	44	—	—	60	44	—	—	—	—	—	—	—	—
Circulation time (sec.); ether (arm to lungs)	20	15	19	20	15	—	—	—	20	—	—	—	—	—	—	—	—
Auricular fibrillation	+	+	+	+	+	+	+	0	+	+	+	+	+	0	0	+	0

The last 5 cases (Nos. 13–17 inclusive) presented marked tricuspid stenosis at autopsy, as did also Case 10. The first 12 cases were carefully studied during life by ourselves.

The cases were comprised of 6 males and 11 females, whose ages ranged from 21 to 60 with an average of 43 years : 4 had no history of rheumatism, and 2 had had chorea only.

liver, palpable spleen, heart enlarged to the left, irregular rhythm, and systolic and diastolic murmurs at the apex. Over the next two years the enlargement of the liver was found constantly.

On examination at present her condition is good. She is able to do her housework and carries on with her sewing work. She has deep and superficial systolic pulsation in the neck veins and is a little cyanosed in lips and finger tips.

The heart is enlarged by X-ray to both right and left, but the lung hilar shadows are not strikingly abnormal. The heart is "mitral" in shape. The œsophagus runs down the front of the vertebral column. On auscultation there are systolic and mid-diastolic murmurs at the apex, and localized systolic and faint diastolic murmurs, separate and distinct, at the lower end of the sternum. The sounds at the aortic area are quite clear. The liver is enlarged two fingers' breadth below the costal margin, and pulsates with systole. B.P., 140/80 mm. EC., auricular fibrillation, no abnormal axis deviation, and digitalis T waves. Venous pressure 21–24 cm. water. Circulation time: ether 15 seconds (normal 5–10); lobeline over 30 seconds (normal 15–20).*

Case 3, male, aged 40 years. First seen by us at the age of 21, following rejection for life insurance. He had run as a member of his high school team and had been apparently quite healthy until one year previously when he had some mild rheumatic pains and some paroxysms of tachycardia. At that time he had a normal sized heart, a blood pressure of 90/65, an accentuated and reduplicated pulmonary second sound, a blowing watery systolic murmur nearer the sternum than the apex, and a short early diastolic murmur in the second and third left interspaces, while the EC. showed normal rhythm with probable early intraventricular block. For the next ten years he was fairly well, working hard, until in 1931 at the age of 32 he developed auricular fibrillation and congestive failure. He made a good recovery, and in 1934 visible venous pulsation in the neck, an enlarged and pulsating liver, and a very large heart were found; in spite of this he was able to walk five miles a day without undue discomfort. The diagnosis of tricuspid stenosis was then queried. In 1935 a note was made that the mid-diastolic murmur was louder at the lower end of the sternum than at the apex. At the end of 1936 ascites began to be a prominent symptom and necessitated paracentesis every ten to fourteen days, 10–12 litres being withdrawn each time. In 1937 his activities were extremely limited by fatigue and dyspnœa, and a prognosis of only a few months of life was given. However, over the past two years his condition has not grown any worse and at present he spends his time resting, walking about the house, and going out for car rides.

On examination his facial appearance is evident in Fig. 2. He shows slight cyanosis, emaciated musculature, a prominent abdomen, and marked systolic, deep jugular pulsation in the neck veins (Fig. 2 A and B); on auscultation there are systolic and diastolic murmurs at the apex, and at the lower end of the sternum there are two other distinct murmurs; the liver is enlarged and pulsating, and there is marked ascites. B.P. 100/70. X-ray examinations, over the years from 1931 to the present, have revealed a steadily increasing heart size, so that now the shadow of the right heart border touches that of the right chest wall. The œsophagus is deviated slightly to the left in the lower half of its course through the thorax. EC., auricular fibrillation and intraventricular block of the right branch type. Vital capacity in 1931, 3550 c.c.; in 1939, only 1900 c.c. The venous pressure is 23·5–24·5 cm. water. Circulation rate: ether time 19 seconds (normal 5–10).

Case 4, male, aged 54 years. Rheumatic fever at the age of 34, in bed 14 weeks. Worked steadily thereafter as a mechanic. Examined after a transient attack of blurring of vision and weakness, there was slight cyanosis, systolic deep jugular pulsation, a slightly enlarged liver with definite expansile pulsation, localized systolic and diastolic murmurs at the lower end of the sternum, and in addition the murmurs diagnostic of mitral and aortic stenosis and regurgitation, and auricular fibrillation. X-ray showed enlargement of the heart to right and left. He is still working as a mechanic.

* For later notes and autopsy see Addendum on p. 165.

Stenosis and regurgitation of the mitral, aortic, and tricuspid valves was diagnosed. Venous pressure was 22 cm. water. Circulation rates: ether time, 20 seconds; saccharin time, 45 seconds. A point of special interest in this case, besides that of his relatively good condition, is that his systolic jugular pulsation was mistaken for a carotid pulse in the medical clinic.

Case 5, female, aged 60 years. Rheumatic fever at the age of 40. She has had twelve children and two miscarriages. She was first seen for her heart trouble because of attacks of tachycardia and fibrillation at the age of 45. First admitted to hospital for congestive failure at the onset of permanent fibrillation ten years ago, at the age of 50 years. At that time she had an enlarged and pulsating liver and ascites and one of us questioned the diagnosis of tricuspid stenosis. For the past ten years she has been much the same, becoming dyspnœic on slight exertion and presenting on X-ray examination a heart enlarged both to right and left, and on physical examination an enlarged and pulsating liver. She lives alone on a hill and is able to do her housework and some shopping and comes to the cardiac clinic once a month.

At present she is slightly cyanosed with systolic pulsation of the neck veins, which has been present for at least one year. Although the liver is large and pulsating, there is no ascites or œdema of the ankles. There is no localized tricuspid murmur. EC., auricular fibrillation and intraventricular block. B.P., 235/100. A diagnosis of mitral and aortic stenosis and regurgitation, tricuspid regurgitation and probable tricuspid stenosis, and hypertension has been made. Venous pressure 21 cm. water. Circulation time: ether, 15 seconds; saccharin, 44 seconds.

Case 6, female, aged 36. First attack of rheumatic fever at the age of 24 and a second attack two years later. She was admitted to the hospital in congestive failure in 1933 and for this a total thyroidectomy was performed. Since then she has been troubled with symptoms of myxœdema and hypo-parathyroidism, and was always on the edge of congestive failure with repeated attacks of ascites and always with an enlarged pulsating liver. A diagnosis of mitral stenosis and regurgitation, tricuspid regurgitation and probable tricuspid stenosis, and aortic stenosis has been made.

Autopsy in January 1941 confirmed the clinical diagnosis of rheumatic heart disease with pancarditis, panvalvulitis, and pericarditis, chronic; cardiac hypertrophy and dilatation; mitral, aortic, and tricuspid stenosis and regurgitation, slight œdema, slight ascites, slight bilateral hydrothorax, pulmonary congestion, and confluent bronchopneumonia of the right upper lobe and right lower lobe.

Case 7, male, aged 57. At the age of 13 years his mother told him that he had valvular heart disease, but he had no definite history of rheumatic fever. His presenting symptoms on admission to the hospital were œdema of the ankles and dyspnœa of three years' duration and abdominal swelling of one year's duration. On the basis of his liver enlargement and ascites, systolic jugular pulsation, enlargement of the heart to right and left, and a localized diastolic murmur, a diagnosis of tricuspid stenosis, in addition to mitral stenosis, was made. He responded well to digitalis, and is now ambulatory and comparatively well though somewhat limited in his activities, twelve months after discharge from hospital.

Case 8, male, aged 50. Rheumatic fever at the age of 17. Otherwise well until two years ago when he had an attack of swelling of the joints, following which he got increasingly short of breath, and his abdomen swelled. A diagnosis of tricuspid stenosis and regurgitation was made on the basis of the history, the marked systolic jugular pulsation, a large pulsating liver that was not tender, ascites, some enlargement of the heart to the right, and a localized systolic murmur at the lower end of the sternum, in addition to the signs of mitral and aortic valve lesions. At present, six months later, he is able to help in the house, do light gardening, and keep his chickens, but he is not able to perform any strenuous work. Venous pressure, 19 cm. water. Circulation rate, using decholin, 60 seconds.

Case 9, female, aged 39. Repeated attacks of polyarthritis from the age of 16 to 34, although she never took any prolonged rest in bed and was doing fairly heavy work

as a housemaid for most of this time. Four years ago admitted to the hospital in congestive failure from which she made a good recovery; systolic jugular pulsation was noted on this occasion. Her last admission was precipitated by excessive exertion on coming to the city every day to go to a sewing centre.

A diagnosis of tricuspid stenosis and regurgitation was made on the basis of the history, the jugular pulsation, a pulsating enlarged liver, a localized systolic murmur at the lower end of the sternum, and X-ray enlargement to the right and left with the œsophagus passing down the front of the vertebral column. The venous pressure was 24 cm. water, and circulation rates were prolonged: with ether, 20 seconds, and with saccharin, 44 seconds. She is ambulatory, but restricted in her activities.

Case 10, female, aged 34 years. Rheumatic fever at the age of 7 and again at 13 with pericarditis. In bed for two years from the age of 20 to 22, and then able to work for eleven years as a stenographer. She was admitted to the hospital with recurrent rheumatism, auricular fibrillation, and congestive failure, twenty months before death. Ascites and œdema and the enlarged pulsating liver showed little variation except for temporarily yielding to diuretics. On account of the persistence of the failure and the pulsation of the liver and in the neck, and a localized diastolic murmur at the lower end and to the left of the sternum, a diagnosis of tricuspid stenosis in addition to mitral and aortic valve disease was made. She died suddenly.

Autopsy showed marked mitral, tricuspid, and aortic stenosis.

Case 11, male, aged 58. Well and active as a youth. Rheumatic fever, at the age of 37, kept him out of work for 4 years, and affected his heart. He felt well, however, thereafter for 13 years. He has had dyspnœa for 4 years and swelling of his abdomen, for the past 2 years, for which he has had to have abdominal paracentesis every few weeks.

A diagnosis of tricuspid stenosis in addition to mitral stenosis was made on the basis of the history of systolic jugular pulsation, an enlarged liver with ascites, a large heart, and localized systolic and diastolic murmurs at the lower end of the sternum.

Case 12, female, aged 29. Rheumatic fever with joint pains; at the age of 7, chorea for six weeks, recurring each winter until the age of 13 when a heart lesion was first diagnosed. Married at the age of 18, there was a normal pregnancy and delivery at 22. The auricles began to fibrillate at 23 and she developed congestive failure. Since then marked limitation of activities, with three attacks of congestive failure and probable rheumatic fever. For the past ten months she has had ascites and œdema of the legs and relatively little dyspnœa, and has been living a bed-and-chair existence.

A diagnosis of tricuspid stenosis has been made upon the history suggestive of long-standing right heart failure, a large pulsating liver, a localized diastolic murmur at the lower end of the sternum, marked enlargement of the heart to the right by X-ray, and slight systolic deep jugular pulsation.

Case 13, male, aged 21, negro. Rheumatic fever at the age of 6; no recurrences. His activity was not restricted. He played games, graduated from high school, and worked as a day labourer until he developed acute rheumatic fever two months previous to his admission to hospital. He was admitted with tremendous œdema of the legs and abdomen. Fibrillation of the auricles was present.

Autopsy showed marked stenosis of mitral, aortic, and tricuspid valves.

Case 14, female, aged 41. Chorea at 12 and 14. She married and had five children, the last at the age of 24 years without any difficulties. At 34, she was admitted to the hospital owing to increasing shortness of breath and attacks of pulmonary œdema and a three months' pregnancy which was terminated. X-ray showed enlargement of the heart to the right. The cardiac rhythm was regular. She was able to do heavy housework with the aid of full digitalization, until two weeks before her death from cerebral embolism.

Autopsy showed chronic rheumatic endocarditis, with marked stenosis of both mitral and tricuspid valves, cardiac hypertrophy, embolism and thrombosis to the bifurcation of the abdominal aorta, both external and internal iliac, and both renal arteries, embolus at bifurcation of the basilar artery with occlusion of postero-median

ganglionic branches, infarction of the spleen and both kidneys, pulmonary congestion, and cholelithiasis.

Case 15, female, aged 42. Whooping cough at 5, pneumonia at 9 and 12, and also " rheumatism " in childhood. She had suffered from ill health most of her life, with cough, sputum, and hemoptysis, thought to be due entirely to bronchiectasis. Her third hospital admission found her with marked congestion (anasarca); she died at this time.

Autopsy showed tightly stenosed mitral and tricuspid valves.

Case 16, female, aged 30. At 16, because a rapid regular cardiac impulse was noticeable on the chest wall, she was put to bed for one month, although she had no symptoms at all. After this she resumed her usual active life. At the age of 22 she had paroxysmal tachycardia but was able to play tennis and dance energetically. Two and a half years before death she developed generalized œdema of the extremities, chest, back, and face, with ascites, for which she was bedridden. She was not relieved by diuretin or digitalis.

Autopsy revealed mitral and tricuspid stenosis and active rheumatism.

Case 17, female, aged 41. Chorea at 12 and 14, and a syncopal attack at 17, following which valvular disease was discovered. At 27 she collapsed while nursing and from then onwards she was much limited by dyspnœa. Ten years ago (at 31) œdema of her ankles set in and marked cyanosis appeared two years ago. Following her mother's death she became psychopathic and was admitted to the hospital. At this time her lungs were clear, but she showed a slightly enlarged liver, ascites, and slight œdema of the legs. She grew worse and died.

Autopsy showed marked stenosis of aortic, mitral, and tricuspid valves, the tricuspid valve barely admitting the tip of the finger.

Discussion

Tricuspid regurgitation as evidenced by pulsating neck veins, a distended and pulsating liver, tricuspid murmurs, and in some cases by the absence of any respiratory distress in spite of marked ascites and œdema, is not uncommon in children suffering from rheumatic fever. The signs may persist for some months and only disappear with the disappearance of the rheumatic fever and the recovery of normal function of the heart muscle. It is therefore unwise, save in exceptional cases, to make a diagnosis of tricuspid valve disease in the first two decades of life, or in a patient within five or six years of the onset of his rheumatic fever.

Cases presenting the full tricuspid syndrome of ascites, œdema, pulsating neck veins, and surprising functional ability have been reported, in whom autopsy has revealed only an irreversible stretching of the valve and no endocarditis (Fischer, 1928, Holzman, 1932, and White and Cooke, 1939). There appears to be no method of avoiding this diagnostic pitfall. The stretching of the tricuspid ring, which occurs during failure but may persist even after the right ventricle has recovered in large part or wholly, presents the chief diagnostic difficulty. This stretching of the valve ring may be present in a case of isolated mitral stenosis, in which instance the full tricuspid syndrome will be simulated.

Some cases of long-standing systemic arterial hypertension develop this " tricuspid syndrome " over the last three to four years of their life. At autopsy the only abnormality is some dilatation of the tricuspid valve ring with the production of incompetence.

Finally, of course, cases of constrictive pericarditis must be distinguished. The absence of heart murmurs and of any more than the slightest enlargement of the heart should be sufficient to distinguish these cases from rheumatic heart disease.

For interest we determined the average age at death of 160 consecutive autopsied cases of rheumatic heart disease, excluding those with tricuspid involvement. This proved to be 43·5 years. The same determination was carried out in the series of 30 autopsied cases of tricuspid valve disease referred to earlier in this paper with the finding of 23·4 years; however, the average in our series of 17 cases whose histories we have presented was 43 years, with several patients still alive.

Gross, Kugel, and Rothschild (1934) in careful investigations showed that in the first five decades of life, lesions indicative of active rheumatism were present in over 80 per cent of cases with rheumatic heart disease dying of congestive failure. As patients grow older the frequency of attacks of rheumatic fever tends to grow less. In the young age group the attacks are frequent. In the first three decades, therefore, it is probably true to say that it is the state of the myocardium that plays the dominant part in the survival of the patient, while afterwards, the mechanical factors produced by the valve lesions play an increasingly important role in determining whether the heart can continue to function adequately. The whole group can consequently be divided into two, a young group in the first three decades of life and an old group. In the young group the greatest number of autopsies will occur in a general hospital as the patients are often acutely ill with rheumatic fever. The more severe the infection, the more likelihood there is that the tricuspid valve is affected. If, therefore, one can make a diagnosis of tricuspid stenosis in this first group, it indicates an extremely grave prognosis as to future life.

In the older group, the mechanical factors produced by the valve deformity begin to play an important role. The safety valve function has been recognized for many years, acting either by regurgitation or stenosis in preventing the dangerous distention of the right ventricle or of the lung capillaries that attends marked mitral stenosis. This function is very well illustrated by *Case* 1, a patient who was able to carry on with her household tasks in spite of such marked mitral and tricuspid stenosis as is shown in the illustration (Fig. 4 A and B). Her venous pressure was 21 cm. of water, and she always insisted on sleeping with one pillow only, an observation that is not in keeping with Altschule and Blumgart's contention that orthopnœa is closely related to a raised venous pressure.

The importance of recognizing tricuspid valve disease in this older group is, again, but for a different reason, in establishing the correct prognosis. As has already been stressed, these patients, although they will probably die at an earlier age than similar patients without the tricuspid valve affected, are able for some years to live a sheltered and useful life, although constantly presenting signs and symptoms that would lead one to expect a much earlier demise.

Thus, although well-marked tricuspid valve disease (stenosis) is found in

cases of rheumatic heart disease who live somewhat shorter lives than do those without tricuspid valve disease, this lesion is found in those cases who survive the longest (a matter of several years at least, as a rule) after the systemic veins (including the jugulars) and the liver become permanently engorged, with the appearance of ascites.

The realization of the true state of affairs in these older cases indicates the correct line of treatment. The chief complaint is usually recurrent or persistent ascites and œdema. Fluids must therefore be restricted and carefully balanced with the urinary output, a procedure these patients perform for themselves. Mercurial diuretics at regular intervals, or from time to time when required, give good results. Digitalis may or may not be indicated, depending chiefly on the heart rate when auricular fibrillation is present; as a rule it is necessary. Occasionally these measures are not sufficient and then abdominal paracentesis may be carried out when needed. It is unfortunately still true in many places that the auscultation of murmurs indicative of rheumatic heart disease in these patients leads automatically to the administration of large doses of sodium salicylate and sodium bicarbonate. Such drugs in these patients can only increase the patients' discomfort by causing increased water retention. For this reason, also, diets should be kept as low as possible in salt, and frequent saline diuretics such as those frequently advertised in the press for the production of a feeling of " well being " should be avoided.

It will be noted that the cases we are reporting are, with two exceptions, in the fourth, fifth, and sixth decades. The antecedent histories in all these cases are similar—the usual story being: (1) a single attack of rheumatic fever, leaving no demonstrable lesions until some years later when heart disease is discovered on routine examination; (2) attacks of chorea without any apparent heart involvement; or (3) isolated attacks of rheumatic fever in the fourth or fifth decade. In none of these cases was there a period of chronic ill health which is the commonest course for rheumatic fever to follow. In the past histories this group is very similar to a series with so-called " pure " mitral stenosis reported by Walsh, Bland, and Jones (1940). They found that patients who had had a single attack of rheumatic fever in childhood without any residual heart damage or those who had no definite previous rheumatic symptoms frequently appeared later with well marked mitral stenosis. A suggested explanation of this marked stenosis is that there never has been any great dilatation of the heart and that therefore gradual and progressive fibrosis of the valve has been allowed to proceed undisturbed during a long continued active rheumatic infection that has largely passed unnoticed.

Summary and Conclusions

Between 1920 and 1937 there were 217 cases of rheumatic heart disease occurring in 4300 autopsies at the Massachusetts General Hospital. In 47 of these the tricuspid valve was affected, but in only 30 was tricuspid stenosis thought to be of sufficient degree to be of clinical significance.

In addition 12 cases of tricuspid valve stenosis have been examined

clinically by the authors during the past three years including 3 cases that came to autopsy.

There were 21 males and 21 females in the combined groups of 33 autopsied and 9 clinical cases.

The ages at death varied between 10 and 59 years, and the average age for the 30 cases with autopsies was 23 years. The average age at death of 160 cases of rheumatic heart disease in the same hospital was 42 years.

The cases of tricuspid valve disease may be divided into two groups: a young group in the first three decades dying of rheumatic fever, and an older group in whom the mechanical factors induced by the lesions played an increasingly important part.

The symptoms in the younger group were almost indistinguishable from those of rheumatic fever: the older group was characterized by the relatively long survival after the appearance of congestive symptoms and signs indicative in most other circulatory disorders of death in the near future.

The diagnosis of tricuspid disease in the young group indicates serious involvement of the myocardium and a poor prognosis; in the older group owing to the " safety valve " function of the tricuspid valve, the patients may live many years providing there is no recurrence of severe rheumatic fever.

The diagnosis of tricuspid disease is difficult, but when due attention is paid to the history, clinical examination of the patient, and X-ray of the heart, the diagnosis should be made more frequently. No one sign is pathognomonic, but in the order of importance the clues may be listed as follows (their chief value lies in combination): a mid-diastolic murmur localized over the tricuspid area, chronic and well-marked systolic pulsation of the deep jugular veins, ascites in the absence of lung congestion, enlargement of the heart shadow to the right, deviation of the œsophagus to the left, cyanosis and sometimes jaundice, enlarged liver with or without pulsation, persistently raised venous pressure, and prolonged right heart circulation time. The chief reason that the diagnosis is not made more often is that these clues and signs are not looked for.

The diagnosis of tricuspid disease is important as an aid in the more accurate determination of prognosis and treatment.

Addendum

Later Notes of Case 2.

A steady increase of dyspnœa, œdema, and ascites over a period of four months brought her into the hospital again on June 20, 1941, with marked anasarca, and she died two days later. Post-mortem examination showed a moderately hypertrophied heart with marked enlargement of the right ventricle which formed a large portion of the apex of the heart. Weight 450 grams. Right ventricle wall thickness was 5 mm. Mitral valve stenosed, buttonhole type; the circumference was 8 cm.; small thrombus in the left auricular appendage. Aortic valve very slightly involved by rheumatic adherence of the cusps. Aorta smooth. Pulmonary valve normal. Right auricle very large. Tricuspid valve moderately stenosed with shortened chordæ tendineæ. No thrombi in right auricle. Small recent pulmonary embolus and infarct (probably the immediate cause of death). Liver cirrhotic and congested. Kidney congested. Spleen normal.

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IDIOPATHIC CYSTIC MEDIAL NECROSIS OF THE AORTA

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Cystic necrosis of the aortic media is recognized as a cause of dissecting aneurysm and of spontaneous rupture of the aorta. Its occurrence in association with aneurysmal dilatation of the unruptured aorta is sufficiently rare to warrant the following case being reported.

Clinical Report and Necropsy

A man, aged 36, attended the out-patient department of the Middlesex Hospital in February 1940. He had never had rheumatic fever, chorea, or syphilis. Ten years previously he had first noticed dyspnœa on exertion and had been told at the time by his doctor that he had heart disease. During 1938 and 1939 he had become increasingly short of breath on effort, and for several weeks prior to coming to hospital he had suffered from paroxysms of nocturnal dyspnœa with blood-stained expectoration.

On examination he was breathless and orthopnœic. The pulse was regular and collapsing; the brachial arteries normal; the blood pressure 150/60 mm. The apex beat was in the mid-axillary line; systolic pulsation was visible in the second and third right intercostal spaces near the sternum; and systolic and diastolic thrills were palpable in the same area. A loud blowing diastolic murmur was heard on the left of the sternum and at the apex. There were widespread râles throughout both lung fields, but signs of systemic congestion were absent; the cervical veins were not engorged, the liver was not enlarged, and there was no œdema. The Wassermann reaction was negative.

X-ray examination showed gross enlargement of the left ventricle, aneurysmal dilatation of the ascending aorta, and increased aortic pulsation (Fig. 1); there was no radiological evidence of mitral stenosis on screening. An electrocardiogram showed normal rhythm, with left axis deviation, a P–R interval of 0·20 sec., and inversion of the T waves in leads I and II.

The patient died in an attack of acute pulmonary œdema, four days after admission to hospital.

Necropsy.—(Permission was obtained for removal of the heart and lungs

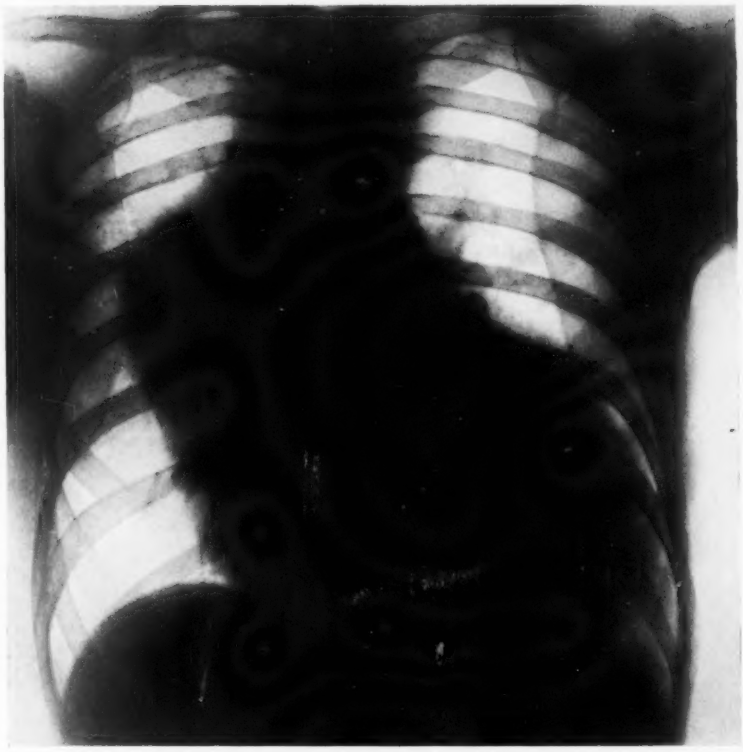

FIG. 1.—Radiograph showing aneurysmal dilatation of the ascending aorta and enlargement of the heart to the left.

only.) The heart weighed 27 oz. (765 g.). The left ventricle was both hypertrophied and dilated; the heart muscle was firm. The aortic ring was dilated, the valve cusps were thin and larger than normal, but appeared competent; the other valves were normal. The ascending aorta as far as the commencement of the innominate artery was uniformly dilated, measuring approximately 6 cm. in diameter (Fig. 2). The wall of the aorta was slightly thinner than normal, and the intima was smooth. The coronary arteries were normal. Histological examination of the aorta showed cystic medial necrosis.

A histological report by Dr. R. W. Scarff, of the Bland Sutton Institute of Pathology, was as follows:—

" The main feature of sections (see Fig. 3), taken both at and below the lesion, is widespread degeneration of both muscular and elastic tissue with the formation of small cystic areas, containing mucoid material, which stains bluish with Mayer's hæmalum but does not give a positive muci-carmine stain. With Van Gieson's stain very little muscle can be found, and there is some fibrous tissue replacement, and no marked increase in the sub-endothelial fibrous

FIG. 2.—Photograph of the heart, post-mortem, showing a greatly dilated ascending aorta.

tissue. With Weigert's elastic stain there seems to be diminution in the elastic material, and complete absence of it in the extensive mucoid areas. In other parts of the section the elastic material appears as irregular short lengths of fibre which are fragmented; no internal or external lamellæ can be distinguished. The appearances are those of intense degeneration of musculo-elastic tissue with cystic areas and areas of fibrous replacement." (See Fig. 3.)

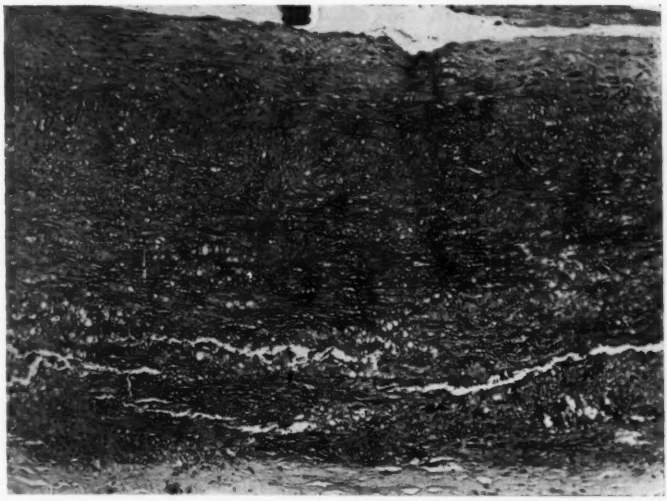

FIG. 3.—Section of the aorta showing areas of degeneration with cystic change. Magnification × 65.

DISCUSSION

The clinical picture was that of terminal left ventricular failure in a man, aged 36, with aortic incompetence and aneurysmal dilatation of the aorta. The radiological appearances were suggestive of a syphilitic ætiology, though the history of heart disease of ten years' duration and the negative Wassermann reaction were against this. Cystic medial necrosis was not suspected.

Rottino (1940) stated that only 2 cases of cystic medial necrosis in unruptured aortas had been reported. Signs of aortic incompetence due to dilatation of the aortic ring have been observed in dissecting aneurysm (Resnik and Keefer, 1925), and a case of medial necrosis was described by Roberts (1939) in a man, aged 33, with dilatation of the aorta, aortic incompetence, and congestive failure; death was due to spontaneous rupture of the aorta. At necropsy, the aortic ring was dilated, and section of the aorta showed medial necrosis, the valves being normal. Rottino (1939) reported the case of a woman, aged 70, in whom the ascending aorta was diffusely dilated and thin-walled, and macroscopically the intima simulated syphilitic aortitis, but there were no murmurs of aortic incompetence. Dilatation of the ascending aorta with aortic incompetence occurred in Harrison's case (1939), but as coarctation was also present it is difficult to assess the part played by medial necrosis in causing the dilatation.

Medial necrosis is a common finding in cases of spontaneous rupture of the aorta. Glendy, Castleman, and White (1937) found it in 8 of 19 cases of ruptured aorta, and Klotz and Simpson (1932) in all of 5 cases, the youngest being a woman, aged 23, who was pregnant. Moritz (1932), examining routine unselected necropsy material, found medial necrosis in 6 of 70 cases, and

Rottino (1939) found an even higher incidence—95 cases of medial degeneration in a total of 210, with cystic changes in 7 cases.

Since the first descriptions of cystic medial necrosis by Gsell (1928) and Erdheim (1929), detailed accounts of the pathology have been given by Moritz (1932) and Rottino (1939, 1940).

The most advanced changes occur in the ascending aorta, especially at its root just above the valves; this is also the site of election for spontaneous rupture. The ætiology of the condition is still unknown. It was first thought to be a degenerative process associated with old age, but subsequent reports have shown that it occurs not infrequently in young people. Toxæmia, nicotine, hyperadrenalism, and hypertension have all been mentioned as factors. It is generally agreed that syphilis plays no part.

It is evident from the reports cited that cystic medial necrosis of the aorta is by no means excessively rare, and that it may cause diffuse dilatation of the ascending aorta and aortic incompetence. It should therefore be considered in the differential diagnosis of unexplained dilatation of the aorta.

Summary

A case of idiopathic cystic medial necrosis of the aorta, without rupture, has been reported. The presenting signs were those of aneurysmal dilatation of the aorta with aortic incompetence and terminal left ventricular failure.

I have to thank Dr. G. E. S. Ward, Lieut.-Colonel Evan Bedford, and Dr. G. E. Beaumont for permission to publish this case, and Dr. R. W. Scarff for the histological report.

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THE ELECTROCARDIOGRAM OF THE STOKES-ADAMS ATTACK

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The combination of a slow pulse with syncopal, epileptiform, or pseudo-apoplectic attacks was briefly mentioned by Morgagni in 1761; then it was more fully described by an Edinburgh physician, Spens (1793), and by Adams (1827) and Burnett (1827), equally well in the same year. Mayo (1838), reviewing examples of slowness of the pulse and quoting Spens, included one such case; and Holberton (1841) added another. Stokes (1846) wrote the important paper that drew general attention to the subject: because of this, and because Huchard (1899) originally proposed the term Stokes-Adams disease, we strongly favour its retention in this order and excluding the use of the word " syndrome ", which would imply that all similar cases whatever the underlying pathology were to be included.

Shortly after Stokes's publication, the discovery was made of the slowing effect on the heart of vagal stimulation, and this governed the views on bradycardia until the end of the last century. Since 1900, with development of our knowledge of heart block, the newer conception placed bradycardia in general as due to an intrinsic myocardial lesion and seldom to a vagal effect. But from this division into neurogenic and myocardial causes another difficulty arises when we consider the definition of Stokes-Adams disease to-day. As our knowledge of the disease is infinitely greater, there is need to define it afresh. Shall it include all cases of cardiac syncope whether these result from vagal action or from ventricular asystole of myocardial origin? We think not, believing that heart block of some grade should be present at some time if the term Stokes-Adams attack is to retain a distinctive meaning. Our reasons are that the great majority of the clinical cases in question are due to myocardial disease with block and that their course, prognosis, and treatment are similar; whereas the reported cases of neurogenic origin (usually without block) are rare and of varied ætiology and prognosis, and may reasonably be classified as cardiac syncope.

Definition.—Stokes-Adams disease is a name applicable to patients with heart block who suffer from recurrent attacks of loss of consciousness due to ventricular standstill, ventricular tachycardia, ventricular fibrillation, or a combination of these.

During a Stokes-Adams attack from ventricular standstill the auricle continues to beat, whereas in other cardiac syncopes as a rule there is total cardiac standstill.

The term cardiac syncope may be reserved for attacks in patients without heart block due to total cardiac standstill from neurogenic or myocardial causes.

Cardiac syncope of neurogenic origin.—An ordinary faint in healthy people is most often of vaso-vagal origin with a fall of blood pressure combined with bradycardia (Cotton & Lewis, 1918), and we have recorded this event in a healthy man of 40, who happened to faint in the cardiographic chair (Fig. 1). In healthy people, too, vagal, carotid sinus, or ocular pressure (Fig. 2) may

FIG. 1.—Syncope in a healthy man. (A) Before ; (B) During faintness; (C) Syncope; (D) Recovery.

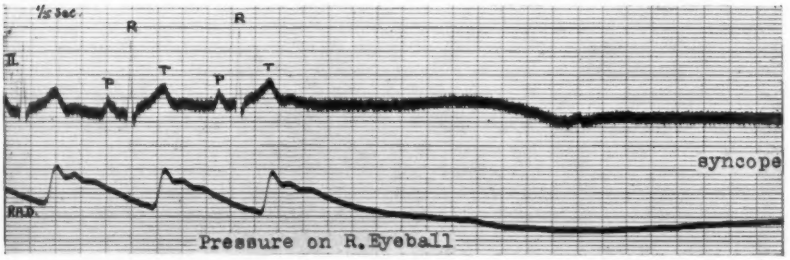

FIG. 2.—Induced syncope in a healthy boy. Pressure on right eyeball.

produce bradycardia and even ventricular standstill with syncope (Lewis, 1925). Transient A-V block may thus be induced (Weiss & Baker, 1933). There are rare but interesting cases of syncope from disease of or near the carotid sinus, or affecting the vagus in its course (Cassidy, 1928; Cassidy & Page, 1928; Gluch, 1932; Weiss & Ferris, 1934; Levy, 1939). A few of

them have been reported as Stokes-Adams attacks, but such cases are not included under our definition, though they are on the borderline, certainly when associated with transient and neurogenic A-V block (Weiss & Ferris, 1934).

Cardiac syncope of myocardial origin.—Paroxysmal ventricular tachycardia (Allan, 1926; Lukl, 1937; Grœdel & Kisch, 1939), the same as a complication of nodal bradycardia (Dressler, 1929) or from severe myocardial damage during a fatal infection (Schwartz & Jezer, 1934), may produce loss of consciousness and approximate to a Stokes-Adams attack. The episodic nature of the attacks, and the absence of heart block distinguish them from it.

In auricular flutter, syncope may be determined by the assumption of a 1: 1 rhythm at the high auricular rate. Fæssler (1939) has described the case of an infant in which nodal bradycardia without standstill produced syncopal attacks.

Incidence of and Varieties of Stokes-Adams Attacks

Only a proportion of patients with heart block get Stokes-Adams attacks. Of Cowan and Ritchie's (1935) 78 cases of complete heart block, one third gave a history of attacks. In Graybiel and White's (1936) series of 72 cases, there were 44. Figures for their incidence in partial or variable block are scarce, but Downie (1929) found Stokes-Adams attacks distinctly more common in partial block than in complete block. It seems likely that a block changing from partial to complete, and a rapidly developing block are periods most susceptible to Stokes-Adams attack, but it is a mistake to assume that with established complete heart block a patient becomes immune from attacks.

It is widely believed that ventricular standstill is the only common disturbance of mechanism which, supervening in heart block, determines the loss of consciousness; and this is implied in many text-book descriptions. But other disturbances of the cardiac mechanism may be responsible for the cerebral attack, and it was with the object of deciding the relative frequency and importance of the mechanisms of the actual Stokes-Adams attack that this investigation was made.

We have restricted our inquiry to cases in which an electrocardiogram was recorded during the unconsciousness of a Stokes-Adams attack. Of these we have 8 at our disposal, and we have collected and studied 56 reported cases. As a consequence we have been led to adopt the following classification :—

Group I.—Ventricular standstill alone.

28 reported cases; and our Cases 1, 2, 3, 4, 5. (Table I).

Group II.—Ventricular tachycardia followed by ventricular standstill.

(*a*) Low ventricular tachycardia (low V.T. in this paper means a rate below 160) followed by ventricular standstill; 3 reported cases and our Case 6. (Table II*a*.)

(*b*) High ventricular tachycardia (high V.T. means in this paper a rate between 200–500), usually also with ventricular fibrillation, followed by ventricular standstill; 13 reported cases and our Case 7. (Table II*b*.)

ABBREVIATIONS USED IN TABLES

A.	=	auricle	H.F.	=	heart failure
A.F.	=	auricular fibrillation	L.B.B.B.	=	left bundle branch block
A.P.	=	angina pectoris	N.R.	=	normal rhythm
B.B.B.	=	bundle branch block	R.B.B.B.	=	right bundle branch block
B.P.	=	blood pressure	Sl.	=	slight
C.H.B.	=	complete heart block	St. Ad.	=	Stokes-Adams attack
C.T.	=	coronary thrombosis	V.	=	ventricle
EC.	=	electrocardiogram	V.F.	=	ventricular fibrillation
Ex.s.	=	extrasystole (ventricular)	V.St.	=	ventricular standstill
H.B.	=	heart block	V.T.	=	ventricular tachycardia

TABLE I

GROUP I.—STOKES-ADAMS ATTACKS DUE TO VENTRICULAR STANDSTILL ALONE

Author	Age	Sex	Clinical features	EC. between attacks	EC. immed. before attack	EC. during attack	EC. immed. after attack	Unconsciousness (duration)	Outcome
Wilson and Robinson (1918)	48	F.	St. Ad. one day	(*a*) C.H.B.; V. 66, A. 110 (*b*) 4:1 and 2:1 H.B. (*c*) N.R. (110) P–R 0·32 sec.	C.H.B.; V. 90, A. 110	V. St., A. to 120	C.H.B. V. 90	(V.St. 7–11 sec.)	Improved
Hay (1921)	—	—	—	—	N.R. 50 to 64, P–R 0·26, B.B.B.	V.St., A. 66–88	—	(V.St. 5·5 sec.)	—
Wiltshire (1923)	64	M.	Slow reg. pulse	C.H.B.	—	V.St., A. rapid then slow; occas. flutter	—	1–1·5 min.	—
Wenckebach and Winterberg (1927) Case 1.	—	—	—	(*a*) N.R., B.B.B. (*b*) partial H.B. (2–5:1)	Normal P–R, then A. rapid; dropped beats	V.St., A. reaches 150	V.T. (140 in one attack)	(V.St. 18–23 sec.)	—

Wenckebach and Winterberg (1927) Case 2.	16	F.	—	(*a*) P–R+ (*b*) Partial H.B., B.B.B.	C.H.B.; V.39, A. 110; occas. 2: 1 H.B.	V.St.	—	(V.St. 11 sec.)	—
Wenckebach and Winterberg (1927) Case 3.	—	M.	St.Ad. weeks; produced on emotion, never on exertion	Variable H.B.	2: 1 then 3: 1 H.B.	V.St., A to 120	—	(V.St. 7·8 sec.)	Died during attack
Stecher (1928)	65	M.	St.Ad. one week	C.H.B.; V. 20–30, A. 60–70	—	V.St., A. 58–76, ectopic P.	3: 1 H.B. P–R 0·36 sec.	10–20 sec. (V. St. 10 sec.)	Attacks ceased
Heimann (1929) Case 1.	60	M.	—	(*a*) C.H.B. (*b*) 2: 1, 3: 1 H.B. (*c*) P–R+; B.B.B.	—	V.St., A. 80 irreg.	—	34 sec.	—
Heimann (1929) Case 2.	—	—	—	—	—	V.St., occas. with A.F.	—	8 sec.	—
Yater and Willius (1929)	74	M.	St.Ad. 3 months. B.P. 240/105	(*a*) 3: 1, 2: 1 H.B. (V. 38; A. 75). (*b*) N.R. 75	C.H.B.; B.B.B. V, 55, irreg., A. 120	V.St., A. 130, then 50, then V.St., no P.	Basic compl. C.H.B.; V.10, A. 100	(V. St. 16–35 sec.)	Died during attack. Necropsy
Cheer and T'ang (1932)	66	M.	St.Ad. 10 months	N.R. 85	C.H.B.	V.St., A. 120	N.R.; P–R 0·18–0·20 sec.	(V.St., 3·6 sec.)	Attacks ceased
Condorelli (1932)	72	M.	—	(*a*) P–R+ (*b*) C.H.B. Partial B.B.B.	C.H.B. 20	V.St.	V.T.	18 sec.	—
Wood (1932)	48	M.	St.Ad. 9 months	C.H.B.; V. 20–30, B.B.B.	—	V.St. A. 75–86	—	2–3 min. (V. St., 7·5 sec.)	Attacks ceased
Géraudel and Others (1933)	78	F.	B.P. 210/70; uræmia	(*a*) 2: 1 H.B.; V. 30, A. 60; partial B.B.B. (*b*) 1: 1 H.B.; V. 60	H.B.; V. 63, A. 71	V.St., P. quickens	H.B. as preceding	15–40 sec.	Death during attack. Necropsy

TABLE I—*continued.*

Author	Age	Sex	Clinical features	EC. between attacks	EC. immed. before attack	EC. during attack	EC. immed. after attack	Unconsciousness (duration)	Outcome
Pardee (1933)	—	—	—	—	C.H.B.; V. 54, A. 96, R.B.B.B. B. then normal QRS	V.St.	—	(V.St. 15 sec.)	—
Sachs and Traynor (1934)	43	M.	St.Ad. months. Normal heart	N.R. 80	—	V.St. A. 110	C.H.B.; V. irreg., A. 90; R.B.B.B. then N.R.	1 min. (V.St. 5 sec.)	Attacks ceased
Gilchrist (1934)	61	F.	St.Ad. one year	(*a*) 2: 1 H.B. P–R 0·18 sec. (*b*) C.H.B. V. 28, A. 70	2: 1 H.B. P–R 0·19 sec.	V.St.	3: 1 H.B.	(V.St. 10 sec.)	Attacks ceased
Laufer (1934)	68	M.	St.Ad. preceded by angina	(*a*) P–R 0·26–0·32 sec. Partial B.B.B. (*b*) 2: 1, 3: 1 H.B.; C.T. (post.)	Sinus tachycardia. P–R 0·36, dropped beats	V.St. A. 92–142	2: 1, 3: 1 H.B.	(V.St. 11 sec.)	Attacks ceased
Cowan and Ritchie (1935)	49	F.	—	—	—	V.St. (A. 120)	—	(V.St. 11 sec.)	—
Clerc and Levy (1936). Case 1	67	M.	St.Ad.months. H.F.	C.H.B. V. 38; A. 95	—	V.St. occas. P; then V.T.	V.T.100–48 —27	(V.St. 30 sec.)	Died after 4 years (cerebral thromb.)
Schwartz (1936,b)	53	M.	St.Ad. one day, with C.T.	(*a*) C.H.B. (*b*) N.R.	—	V.St. A. 110	C.H.B.; then N.R.	(V.St. 9·5 sec.)	Attacks ceased Necropsy
Hermann and others (1937)	65	M.	St.Ad. 14 years. P. 80, reg.	(*a*) N.R. 75; partial B.B.B. (*b*) C.H.B. only day before death.	N.R. A. 100; dropped beats	V.St. A. 110–50 Ectopic P.	Nodal (A–V) rhythm, then N.R. also V.T.	(V.St. 45–150 sec.)	Died in observed attack Necropsy

Norris and Landis (1938). Case 1	—	—	—	—	—	V.St., variable P.	—	(V.St. 4 sec.)	—
Cossio (1939)	—	—	—	—	C.H.B.; V. 34, A. 105	V.St., A. to 120	—	(V.St. 8·5 sec.)	—
Laplace (1939)	29	M.	—	C.H.B.; V. 50, A. 135	C.H.B.	V.St., P. slow then absent	—	—	Died in observed attack
Sigler (1939, A)	57	M.	A.P.	N.R.; R.B.B.B.	2: 1 H.B. then C.H.B.	V.St., P. 100–116	2: 1 H.B., also N.R.	30–60 sec.	Attacks ceased
Scherf and Boyd (1940)	—	—	H.B. after C.T.	—	C.H.B.	V.St.	C.H.B.	(V.St. 90 sec.)	—
Teran (1941)	51	M.	St.Ad. 4 months. B.P. 190/100;	(*a*) C.H.B.; V. 15 A. 100 (*b*) 2: 1 H.B. R.B.B.B. (*c*) N.R. 66	—	V.St., A. 60–95. Ectopic P.	N.R. 66; P–R 0·22; R.B.B.B.	(V.St. 8–10 sec.)	—

The above Table I comprises 28 reported cases in Group I, to which our Cases 1, 2, 3, 4, 5 also belong.

TABLE II

GROUP II.—VENTRICULAR TACHYCARDIA FOLLOWED BY VENTRICULAR STANDSTILL

(*a*) Low ventricular tachycardia (below 160) followed by ventricular standstill.

Author	Age	Sex	Clinical features	EC. between attacks	EC. immed. before attack	EC. during attack	EC. immed. after attack	Unconsciousness (duration)	Outcome
Gager and Pardee (1925)	59	M.	St.Ad. 2 weeks	(*a*) C.H.B.; V. 27–15; A. 92. (*b*) 2: 1 H.B. (*c*) N.R. 60	V.T. (V. 60; A. 100) then V.St.	V.St. A. 100	—	0·5–1 min. (V.St. 6–20 sec.)	Died after 6 months during attack
Herapath (1926)	63	M.	B.P. 210	(*a*) C.H.B.; V. 35; Ex.s. (*b*) C.H.B.; QRS. of the ex.s. type (*c*) 2: 1 H.B. L.B.B.B.	V.T. QRS of ex.s. type, rate 62	V.St. A. rate +	—	(V.T. 40 sec. V.St. 12–15 sec.)	No further attacks
Coelho (1932)	—	—	—	—	V.T.	V.St.	—	—	—

(*b*) High ventricular tachycardia (200–500), usually also with ventricular fibrillation, followed by ventricular standstill.

Kerr and Bender (1922)	68	M.	H.F. 4 years	C.H.B.; A.F.; R.B.B.B. V. 30–36	V.T. 180–220	V.F. then V.St.	—	1–2 min.	Recovery from attack.
Hœsslin (1925)	31	F.	Slow pulse	C.H.B.; V. 32–34; Ex.s.	—	V.F. then V.St. P. modified during V.St.	V.T. 132–134; of each group QRS has different shape	—	—
Levine and Matton (1926)	52	F.	—	(*a*) C.H.B.; R.B.B.B. (*b*) 3 months later, inversion of T_2 and T_3	Variable Ex.s.	V.T. (? V.F.) 240; V.St. A. 45	V.T. 140; after 7 min. 70	V.F. 200 sec. V.St. 80 sec.	Attack ceased.
Freundlich (1932)	69	F.	B.P. 250/70; St.Ad. one day, preceded by angina	C.H.B.; V. 48; Ex.s.	Multiple, variable Ex.s. V.T. at increasing rate	Irreg. V.T. (? V.F.) 280; V.St.	T inverted in all leads; coronary R–T	5 min. (V.F. 80 sec.; V.St. 7·5 sec.)	Attacks ceased

Lian and Deparis (1934)	52	F.	St.Ad. 2 years; B.P. 220/90	C.H.B.; V. 35, A. 78; partial B.B.B.	—	V.F. 300; V.T. 200; then basic complexes. Once V.St. A. to 120	C.H.B.	1–1·5 min. (V.St. 12 sec.)	Died during attack
Clerc and Lévy (1936) Case 2	75	F.	St.Ad. 9 years	(*a*) C.H.B.; V. 32; (*b*) N.R., P–R Sl.+, R.B.B.B	—	V.F. (280) then V.St.	V.T. 200 for 7 min., then C.H.B.	6 min. (V.St., 60 sec.)	Alive after 10 months; few attacks
Jezer and Others (1936) Case 1	68	M.	St.Ad. 5 months; H.F.	C.H.B.; 30	V.T. 60–120	V.St., A. up to 165, then V.T.	—	Hours; (V.St. 90–180 sec.)	Died after few hours of V.St. and V.T.
Jezer and Others (1936) Case 2	39	M.	St.Ad. 18 months; B.P. 175/85	C.H.B.; A. 70, V. 25.	V.T. 120 then C.H.B. 25, then V.St.	V.St.	V.T. 75 to 120, then V.St. again	(V.St. 30–60 sec.)	Died in observed attack
Spühler (1936)	51	F.	St.Ad. 4 months; A.P. 16 months	(*a*) C.H.B.; B.B.B., (*b*) 2: 1 H.B.; B.B.B.	C.H.B.,B.B.B., multiform Ex.s.; A. 125	V.T. (270). V.St., A. 40, irreg.	V.T.(130); then C.H.B.; V55, A. 170, with normal QRS	2–8 min. (V.T. 115 sec.; V.St. 8 sec.)	—
Froment and Gonin (1938, A)	60	M.	St.Ad. 10 years; H.F.	C.H.B.; V. 13, A. 35; L.B.B.B.	V.T. 60.	V.St. P. later disappears. Occasional V.T. 300	V.T. 40–48–28, then V.St.: 4 hours alternate V.St. and V.T.	(V.St. 30 sec.)	Died in V.F.
Gertz and Others (1938)	59	F.	St.Ad. years	(*a*) N.R. (*b*) 2: 1 H.B.	C.H.B. Ex.s.	V.T. 250 and V.F.; V.St.	C.H.B.	(V.F. 83 sec. V.St. 50 sec.)	Died in observed attack
Norris and Landis (1938). Case 2	—	—	—	C.H.B.	—	V.T. 160, V.St. (A. 100); V.F. 150–200	—	20 min.	Died after two days
Soulié (1938)	68	F.	St.Ad. 2 years, following C.T.	2: 1 H.B.; L.B.B.B.	2: 1 H.B.; C.H.B.	V.St. absent P. V.T. for 600–800 sec. then again V.St.	Multiform Ex.s. C.H.B. with aur. flutter	(V.St. 70–130 sec.)	Died in observed attack

The above Table II, comprises (*a*) 3 reported cases in Group II (*a*), to which our own Case 6 also belongs; and (*b*) 13 reported cases in Group II (*b*), to which our own Case 7 also belongs.

TABLE III

GROUP III.—HIGH VENTRICULAR TACHYCARDIA, OR VENTRICULAR FIBRILLATION, OR BOTH. NO VENTRICULAR STANDSTILL

Author	Age	Sex	Clinical features	EC. between attacks	EC. immed. before attack	EC. during attack	EC. immed. after attack	Unconsciousness (duration)	Outcome
Gaillard (1923)	62	M.	St.Ad.; chronic nephr., B.P.+	C.H.B. with A.F. (V. 40)	—	V.T. (200)	V.T. at low rate	—	Died after 1 month
Gallavardin and Bérard (1924)	55	F.	St.Ad. 4 years	C.H.B.; V. 35–42, A. 85	—	V.F. (336)	C.H.B.	2·5 min.	—
Heimann (1929) Case 3	46	F.	H.F., B.P.+	Partial H.B.	—	V.F. (? V.T.) 200	—	10 sec.	Died during attack
Gallavardin and Froment (1931)	39	M.	Subacute bact. endocarditis; St.Ad. 5 days before death	C.H.B. (40)	N.R. then 3: 1, 2: 1 H.B., partial B.B.B., then V.T.	V.T.	—	—	Died after 5 days
Schwartz and Jezer (1932)	65	F.	St.A. 5 months; H.F.	C.H.B.; V. 28, A. 66; QRS. normal or B.B.B.	Multiple Ex.s.	V.T. and V.F., rate 250–500	Low V.T. to 90, then basic rhythm	10–180 sec.	—
Bizzozero (1934)	60	F.	Diabetes; St.Ad. 1 month. H.F.	C.H.B.; V. 40, A. 110; partial B.B.B.	—	V.F. (and V.T.) 230	—	—	—
Schwartz and Hauswirth (1934)	56	F.	St.Ad. 6 months; B.P. 230/120	(*a*) Sinus tachyc., normal P–R, R.B.B.B. (*b*) 3: 2 H.B. (*c*) C.H.B.	Multiple ex.s.	V.T. and V.F. 250–300	—	Several min.	Died after 1 year with syncope
Kahall (1935)	68	M.	St.Ad. 3 days; B.P. 200/100; P. 34–40	—	C.H.B. multiple ex.s.	V.T.; V.F.; or V.T. only	C.H.B., slow ventr. rate	20–120 sec.	Died after 6 weeks in attack.

Sigler (1938)	58	M.	St.Ad. months; A.P.	Normal P–R, 3: 2 H.B.	As between attacks; runs of Ex.s.	V.T. 220–250; V.F. 370–195	C.H.B.; after 300 sec. V.T. 130	1–2 min.	Attacks ceased
Turrey and Leamann (1929)	—	—	—	C.H.B.	Multiform Ex.s. in runs	V.T. (240), V.F., V.T. with occas. V.St. and absent P.	V.T., C.H.B., Ex.s.	(V.F. 42 sec.)	—
Sigler (1939, B)	66	F.	Diabetes, C.T.; 6 months later St.Ad.	C.H.B.	—	V.T. (250), V.F. (370), V.T.	—	5 hours intermittently	—
Fishberg (1940)	—	—	—	C.H.B.	C.H.B., multiform Ex.s.	V.T. 200–300, then V.F.	—	(V.F. 19 sec.)	—

The above Table III comprises 12 reported cases in Group III, to which our own Case 8 also belongs.

Group III.—High ventricular tachycardia, or ventricular fibrillation, or both, without ventricular standstill.

12 reported cases and our Case 8. (Table III).

Group IV.—Extreme bradycardia with complete heart block.

No table is given for this group because it is small and less important. Many early reports tell of extreme slowness of the pulse as the sufficient cause of the faint, yet electrocardiograms of such cases are hard to find. That of Gilchrist (1934) is an example, yet that was the result of stoppage of ephedrine therapy. The case of Dubbs (1938) is a clear example though it is surprising that the ventricular rate was not lower than 20.

Authors' Case Reports

Case 1, *male, aged* 54.

History.—Three years ago and six months ago he lost consciousness. Recently the attacks became frequent and he was admitted to hospital, where he died four days later during an attack. There was no history of rheumatic fever, chorea, or diphtheria.

Examination.—The pulse was regular and 30 a minute, but next day it fell to 16, persisting at this rate between the attacks until he died. There was no clinical evidence of cardiac enlargement and there were no murmurs. Signs of heart failure, including distension of the liver and œdema of the ankles, appeared on the third day in hospital. The blood pressure (B.P.) was 120/65. The Wassermann reaction (W.R.) was negative.

Course.—He was seen in several Stokes-Adams attacks which occurred about every hour during the last two days of life. A momentary feeling of faintness preceded unconsciousness, then the respiratory movements became exaggerated and profuse sweating accompanied a great pallor. The pupils dilated ; the upper limbs twitched. With the onset of unconsciousness the pulse stopped and the ventricles ceased to beat (auscultation)—for 25 seconds in one attack. When the pulse returned, it was regular at 16 and consciousness was quickly regained. Occasionally the heart sounds were absent for periods over 10 seconds without complete loss of consciousness. Vomiting commonly followed the attacks, but there was no incontinence of urine.

On one occasion, adrenalin (3 minims of 1 in 1000 solution intravenously) quickened the pulse to 72 within one minute, but two minutes later it was 32, and 30 minutes later, 16. On another occasion, two successive injections (5 minims subcutaneously) had no effect on the pulse rate.

Electrocardiographic features

(*a*) *Between* the attacks there was complete heart block (C.H.B.) with a regular ventricular rate of 33 to 25 and an auricular rate of 100. The ventricular complexes were of right bundle branch block (B.B.B.) type (QRS, 0·16 sec.) with deep inversion of T_2 and T_3.

(*b*) *During* the attack (Fig. 3) ventricular standstill was recorded for 3·8 seconds with an increased auricular rate of 150.

FIG. 3.—Case 1. Stokes-Adams attack. Ventricular standstill. Complete heart block.

Case 2, *male*, *aged* 76.

History.—Some heart affection had been recognized for years. Fainting attacks, some with convulsions, occurred for the first time on the day before admission to hospital.

Examination.—The pulse was regular, owing partly to extrasystoles; the rate varied between 30 and 72. The heart sounds were normal, and on X-ray examination the heart was of normal size and the aorta was unfolded. There was no evidence of heart failure, nor of any other than cardiovascular disease. B.P., 180/80.

Course.—In hospital the attacks were at first frequent, sometimes 3–4 a day and each lasting about 30 seconds. He lost consciousness suddenly and his breathing became stertorous; no pulse could be felt and no heart sounds could be heard. He had oral treatment with ephedrine and thyroid, and injection treatment with adrenalin. Gradually he improved and there were no attacks during the last weeks in hospital. He became quite well, and when last seen, four years later, he was still free from attacks.

Electrocardiographic features

(*a*) *Between* the attacks there was at first partial or complete heart block with a ventricular rate of 50–75, apart from multiform extrasystoles. The basic complexes were of supraventricular type (QRS, 0·07 sec.). Fig. 4 was taken at this period. Three days after admission, sinus rhythm returned with a P–R interval of 0·3 sec. The successive records taken in hospital and a fortnight after his discharge were similar. Five months after his discharge, partial heart block was recorded with an irregular ventricular rate of 50–63 and a regular auricular rate of 80. Sinus rhythm with prolonged P–R interval returned one month later, and the last record taken two and a half years later was similar, with only a widened ventricular complex (QRS, 0·12 sec.) of partial bundle branch block appearance.

(*b*) *During* the attack (Fig. 4) there was ventricular standstill recorded during 4 seconds, during which the auricular rate increased from 66 to 86. At the end of the standstill there was partial heart block.

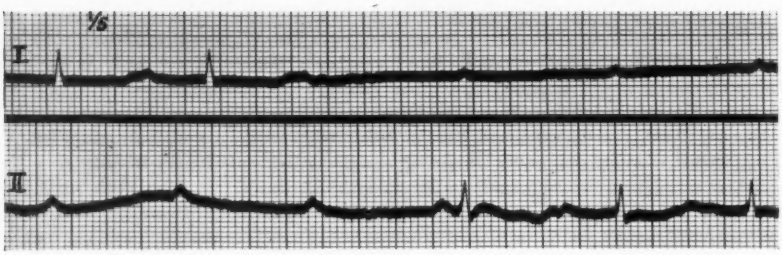

FIG. 4.—Case 2. Stokes-Adams attack. Ventricular standstill. Partial heart block.

Case 3, *male, aged* 68.

History.—For years had been treated for high blood pressure. For one year, recurrent attacks of anginal pain. A few days before examination had attacks of giddiness, sometimes followed by loss of consciousness. The day before examination the syncopal attacks became very frequent.

Examination.—The blood pressure was raised; there were no signs of failure. While being examined, he fainted repeatedly. The colour of the face alternated between pallor and flushing, the breathing became irregular and sometimes ceased for seconds. The slow, regular pulse of about 50 stopped suddenly for some seconds, the heart sounds were inaudible and the patient fell back unconscious. Even when the pulse returned, he was mentally clouded the whole day. He became worse, and died a few days later.

Electrocardiographic features.

(*a*) *Between* the attacks there was complete heart block with a ventricular rate of 57, and an auricular rate of 80, both regular. The basic ventricular complexes were supra-ventricular (QRS, 0·08 sec.) with an inverted T_1.

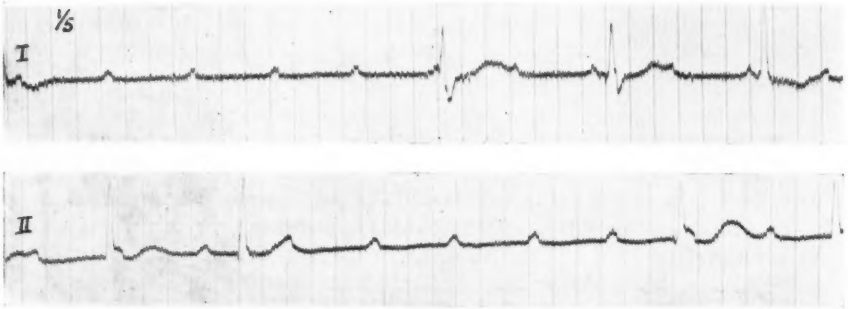

FIG. 5.—Case 3. Stokes-Adams attack. Ventricular standstill. Complete heart block.

(*b*) *During* the attacks (Fig. 5) there was ventricular standstill recorded during 4 and 3·6 seconds respectively. The first ended with an ectopic ventricular complex followed after 1·4 seconds by a similar one. The second attack ended with two basic ventricular complexes at a rate of 46 a minute, but from the third complex onwards the rate was again 57. At the end of the first and longer standstill the auricular rate slightly increased to 92, and the original rate

of 80 was regained only 5 seconds after the appearance of the basic ventricular complex. The second and shorter ventricular standstill left the original auricular rate unaltered.

Case 4, *male, aged* 43.

History. The first attack of unconsciousness happened 14 months ago. No attacks for 4 months, then they became frequent. He was admitted to hospital for observation as epilepsy was suspected. There was no history of acute rheumatism, chorea, or diphtheria.

Examination.—The pulse was regular, 70 a minute. The heart was not enlarged and there were no murmurs; other systems showed no abnormal signs. B.P., 140/80; W.R., negative. During a stay of 38 days in hospital no fewer than 590 attacks were noted. In many he remained fully conscious and complained of a burning sensation over the stomach which spread upwards to the chest, to the neck and face, and sometimes along the arms to the fingers and down the legs to the toes. During such paroxysms he expected to faint, but only turned pale and breathed rapidly. In some of them the pulse stopped and the ventricles ceased beating (auscultation) for periods of 5 seconds or more, jugular pulsation (auricular contraction) continuing. This ventricular standstill was often recorded by electrocardiograph. In almost as many attacks consciousness was lost, and then ventricular standstill lasted 10 to 20 seconds, the patient becoming unconscious towards the middle of the period, the breathing stertorous, and the face muscles twitching.

Course.—The attacks gradually became less frequent, and a month after discharge from hospital they ceased. Later he reported none for three years, and was able to do light work.

Electrocardiographic features. In all, 15 electrocardiograms were taken and they showed that the block was paroxysmal.

(*a*) *Between* the attacks there was most often sinus rhythm at a rate of 85, with a normal P–R interval and normal ventricular complexes (Fig. 6A). On

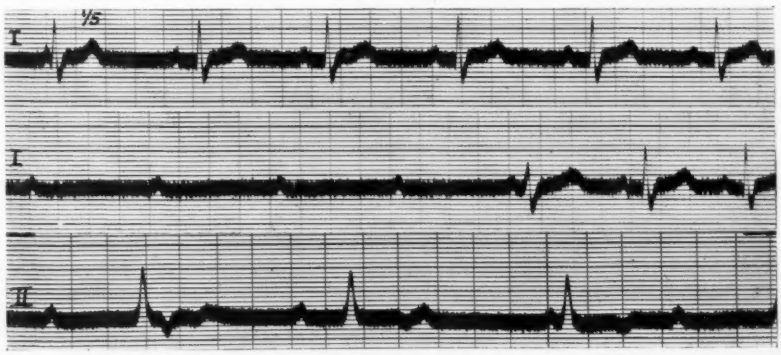

FIG. 6.—Case 4. Stokes-Adams attack. Paroxysmal heart block. (A) Sinus rhythm, normal P–R. (B) Ventricular standstill ending with sinus rhythm. (C) Complete heart block. (B) was taken immediately after (A); (C) on another day.

other occasions complete heart block was recorded with a ventricular rate of 55 and an auricular rate of 90 (Fig. 6C).

(*b*) *During* the attack there was ventricular standstill recorded during 3 seconds. It began abruptly during sinus rhythm with a delayed auricular beat not followed by the ventricular complex (not shown), and ended with an ectopic beat 0·10 second after P, when the sinus rhythm continued at the usual rate of 85 (Fig. 6B). During the standstill the auricular rate increased from 66 to 75.

Case 5, *male, aged* 70.

History.—No complaints until two and a half years before, when he suddenly fell down unconscious in the street. Occasional short faints followed at varying intervals, and for one week as frequently as every day. The day before admission, the attack was more severe; he became extremely pale, and unconscious, and seemed about to die. For months he had noticed dyspnœa and palpitation.

Examination.—He was pale, and every few minutes fainted. The pulse was irregular, about 24 a minute; there was slight enlargement of the left ventricle, and moderate uniform widening of the aortic shadow on radioscopy. The heart sounds were normal; there was no evidence of failure. B.P., 190/75; W.R., negative. The urine contained a trace of albumin.

Course.—Adrenalin (5 minims subcutaneously) did not increase the ventricular rate and did not prevent the recurrence of attacks. During the following 5 months he fainted about once a week, except that on one particular day he had twelve faints. The longest period of freedom was ten days.

Electrocardiographic features.—Polygrams showed complete heart block with frequent pauses of the radial pulse, the longest one recorded being of 8 seconds duration.

(*a*) *Between* attacks, the electrocardiogram showed complete heart block with a regular auricle at 86, and a regular ventricle at 33. The ventricular complexes were of small voltage and of left bundle branch block type (Fig. 7).

(*b*) *During* the attack (Fig. 7), the basic ventricular complex of left bundle

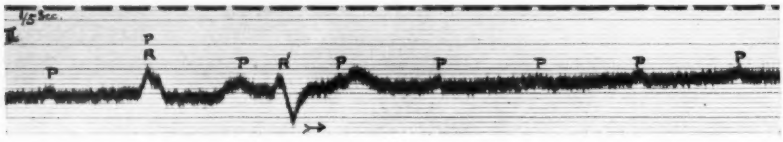

FIG. 7.—Case 5. Stokes-Adams attack. Ventricular standstill. Complete heart block.

branch block type was followed at a distance of 0·7 sec. by a ventricular extrasystole. Then came ventricular standstill, recorded for 2·6 seconds during which the auricular rate was 110. With the reappearance of ventricular action the auricular rate slightly decreased to 100. Ectopic ventricular complexes recurred at irregular intervals.

At the end of another attack (not shown), ventricular extrasystoles were followed after 2 seconds by the returning basic complex. The ventricular rhythm

later increased from 35 to 46 and then changed into a regular ventricular tachycardia at a rate of 60 with widened ventricular complexes of another shape.

Case 6, *male*, *aged* 40.

History.—Nine years dyspnœa, more severe for two years, appearing even on slight effort and compelling him to relinquish heavy work. Seven years, momentary faints (loss of consciousness) about once a month, ascribed to heavy meals or to effort. Ten months ago he had the first severe syncope; its duration was unknown, but there was twitching of face and hands, with incontinence of fæces during the attack, and vomiting after it. Since then he has had up to 100 similar attacks a day, though sometimes he was free for as long as 9 days.

Examination.—The pulse was regular, 28 a minute, and the rate could not be increased by exercise. The apex-beat was forcible but not displaced, and the cardiac dullness was normal. The heart sounds were distant and a faint systolic murmur was heard at the mitral area. The liver was palpable, but there were no other signs suggesting failure. B.P., 125/60 ; W.R., negative.

Course.—While in hospital few days passed without attacks, which were of two different kinds. Some were accompanied by disappearance of the pulse, loss of consciousness, clonic contractions, and incontinence; and they lasted 1–2 minutes. The others, during which he did not lose consciousness, started with a feeling of epigastric oppression, followed by " thumps of the heart ", then by a feeling of pins and needles in the neck which extended over the head. Both types of attack might often recur indifferently over a period of several hours. Adrenalin (5 minims subcutaneously) and barium chloride (0·5 grain, t.i.d.) did not influence the length of the attacks, nor prevent their recurrence. Attacks proved more frequent during a trial course of digitalis (tinct. digitalis. 10 minims, t.i.d.) for seven days. During five months of observation little change was noted and no signs of failure developed. He died in hospital after a succession of attacks in which he complained of epigastric tightness and flatulence. Necropsy was refused.

Electrocardiographic features

(*a*) *Between* the attacks there was always complete heart block with a regular or irregular ventricle at a rate of 20–30. The auricle was fairly regular at 66–86. The basic ventricular complexes were of right bundle branch block type (Fig. 8A). Ectopic ventricular complexes were frequent and varied in shape; they appeared both as extrasystoles following the basic complex, and as regular sequences of a slow ventricular " tachycardia " at 55–75.

(*b*) *During* the attacks several standstills of the ventricle were recorded, the longest lasting 6·8 sec. in which the auricular rate increased from 75 to 100 (Fig. 8B). They were always preceded by a low ventricular tachycardia. Periods with extrasystoles sometimes preceded the ventricular tachycardia.

Case 7, *female*, *aged* 62.

History.—Hypertension was discovered two years before when the patient was in hospital for pulmonary infarction following phlebitis. There was no history of rheumatic fever, chorea, or diphtheria. During the six weeks preceding her present admission she had two Stokes-Adams attacks.

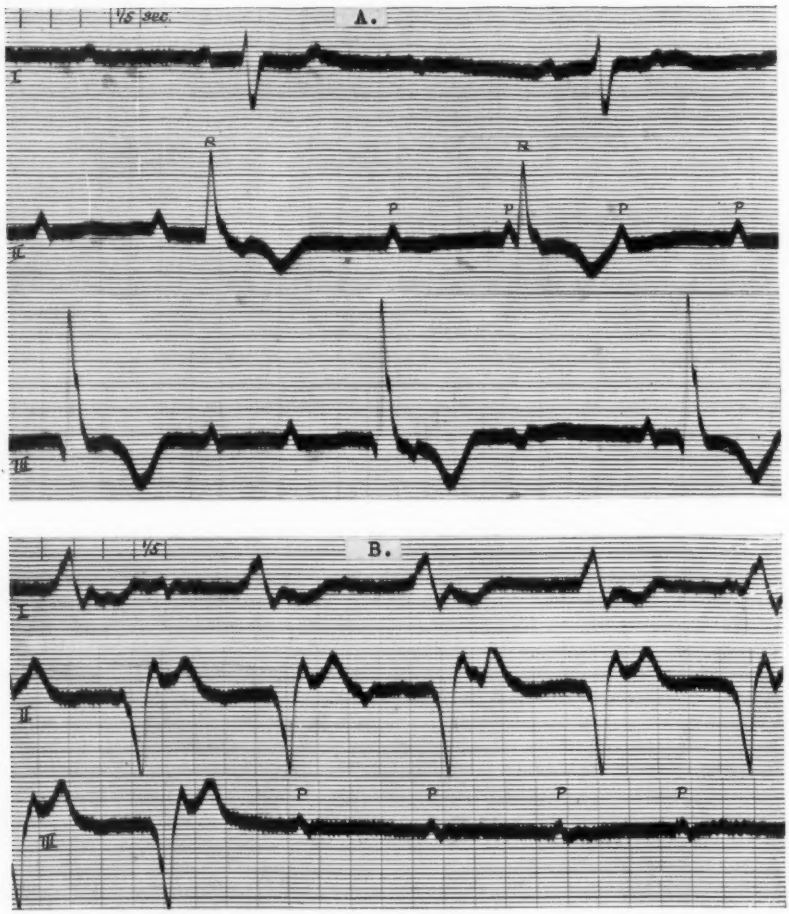

FIG. 8.—Case 6. Stokes-Adams attack. (A) Complete heart block, right bundle branch block. (B) Low ventricular tachycardia preceding ventricular standstill.

Examination.—She looked well and was rather stout. There were no abnormal signs except in the cardiovascular system. As a rule the pulse was regular and infrequent, 38. On radioscopy, the left ventricle was seen to be moderately enlarged. A systolic murmur was heard in both mitral and aortic areas. There was no evidence of heart failure. B.P., 205/110; W.R., negative. Many Stokes-Adams attacks were observed in hospital. On some days there were as many as five, but there might be no attacks for a week or longer.

Clinical features of the attacks.—Often one followed soon after a meal. The patient would complain of feeling suddenly ill and might call for a bed-pan·

Sometimes she would cry out that an attack was coming on. She then became unconscious, the breathing and the pulse stopped, and the heart ceased beating (auscultation). The cyanosed face twitched, and there were spasmodic movements of the arms and legs. The eyes turned up and the pupils dilated widely. The pulse returned after one to two minutes, when it was rapid (about 150 a minute) and usually regular, but later it would fall to about 38 a minute. The breathing, returning soon after the pulse, was stertorous and accompanied by a heaving movement of the chest. Cyanosis gradually disappeared and was usually replaced by slight pallor. After 5 to 15 minutes with returning consciousness she perspired, was exhausted and confused, uttering loud and wild shouts. Incontinence of urine often occurred during an attack.

Electrocardiographic features

(*a*) *Between* the attacks the electrocardiogram changed frequently from complete heart block to 2 : 1 or 3 : 1 heart block when, apart from the pauses, the P–R interval was normal. The ventricular complexes varied from left to right bundle branch block. The ventricular rate was often irregular owing to multiform ventricular extrasystoles. When regular, it varied between 32 and 40 and the auricular rate between 80 and 92.

(*b*) *During* the attack, Fig. 9 was recorded, and the sequence of events is portrayed in Fig. 10 (Chart). Immediately before the attack, the pulse was regular (38 a minute). While the camera was being loaded she lost consciousness suddenly and the breathing ceased. The pulse stopped, but after 30 seconds it returned and the electrocardiogram (Fig. 9A & B) first showed rapid ventricular tachycardia (270 a minute), and the irregular ventricular deflections of *ventricular fibrillation*. This phase lasted 30 seconds when the pulse again disappeared and Fig. 9C & D showed *cardiac standstill* for one minute, and rudimentary P waves 20·8 and 24 seconds after its onset. A subcutaneous injection of 10 minims of 1 in 1000 solution of adrenalin hydrochloride was given at the beginning of the standstill period. The pulse when it returned was regular and infrequent, and the Fig. 9E showed *complete heart block* with an auricular rate of 95 and a ventricular rate of 42. This phase lasted 12 seconds, and then the pulse rose to 135 a minute at the onset of tachycardia with changing ventricular complexes (Fig. 9F) lasting 5 minutes, the auricular rate increasing from 90 to 150 meanwhile. The breathing returned but became slow with long inspiratory and short expiratory phases. At this stage, although still unconscious and not responding to external stimuli, the patient began to utter wild and loud shouts. Fig. 9G showed complete heart block, the ventricular rate was 37 and the auricular rate was as rapid as 150 a minute. The second paroxysm of ventricular tachycardia shown on Fig. 10 (Chart) is not published as it was similar to Fig. 9F. After two minutes, during which the patient ceased to shout and recovered consciousness, the heart rhythm returned as complete heart block at 38. Altogether the patient had been unconscious for nine and a half minutes. We consider it unlikely that adrenalin had any effect upon the sequence of events just described.

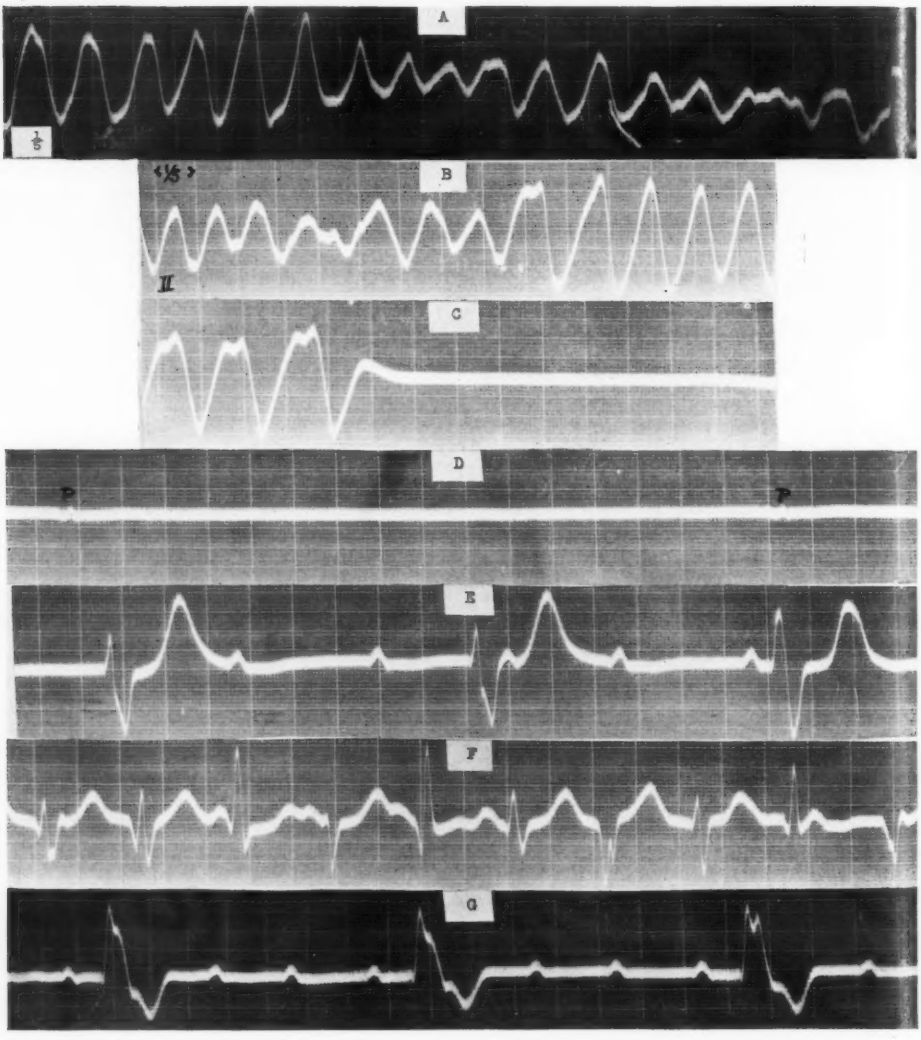

FIG. 9.—Case 7. Stokes-Adams attack. (A) and (B) High ventricular tachycardia and ventricular fibrillation. (C) End of ventricular tachycardia and onset of complete cardiac standstill. (D) Cardiac standstill with two ectopic P waves. (E) Complete heart block, right bundle branch block. (F) Tachycardia with changing ventricular complexes. (G) Complete heart block, left bundle branch block (basic complexes).
All records are lead II.

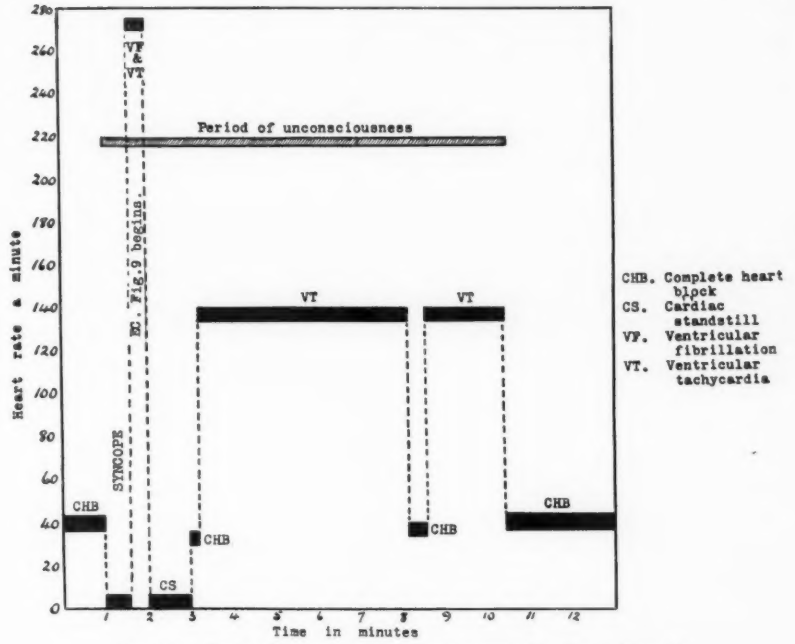

FIG. 10.—Chart of Stokes-Adams attack recorded in Fig. 9 (Case 7).

Case 8, *male, aged* 65.

History.—For seven months angina pectoris, and also dyspnœa on exertion. One week dizzy attacks while sitting. On day of admission he had a bout of dizziness, and later fell unconscious; sent to hospital.

Examination.—Pulse 40, irregular ; no murmurs. B.P. 240/90 ; W.R., negative. Next day an attack was observed; he felt faint, became pulseless, his face pale, then cyanosed and unconscious, then convulsions for two minutes. Respiration ceased, then returned ; he was unconscious from time to time and there were two further convulsions during the next hour. He died one and a half hours after the onset of the attack, during which numerous electrocardiograms were taken.

Necropsy showed a moderate degree of coronary sclerosis with calcification, more of the left than of the right coronary artery. No evidence of myocardial infarction could be found, although there was slight patchy fibrosis.

Electrocardiographic features (Fig. 11)

(*a*) *Between* the attacks there was complete heart block, with an irregular basic ventricular rhythm at 25–32. The auricular rhythm was regular, rate 100. Nearly all the basic complexes were followed at an interval of 0·52 sec. by a standard form of extrasystole with the main deflection downward. This was often followed by one or more different extrasystoles, and then the succeeding basic complex was of course delayed; hence the irregularity of the basic ventricular rhythm.

(*b*) *During* the attack, ventricular tachycardia lasted 6 seconds, and comprised in order : 6 downwards complexes (rate 200 approx.), 2 diphasic complexes, 6 upright complexes (rate 200–210), and 4 downward complexes (rate 200–120) approximating in form to the standard extrasystole. The pause after the tachycardia was no longer than the usual pause after an extrasystole, being about 1·2 seconds. This was confirmed by a short record of the end of another

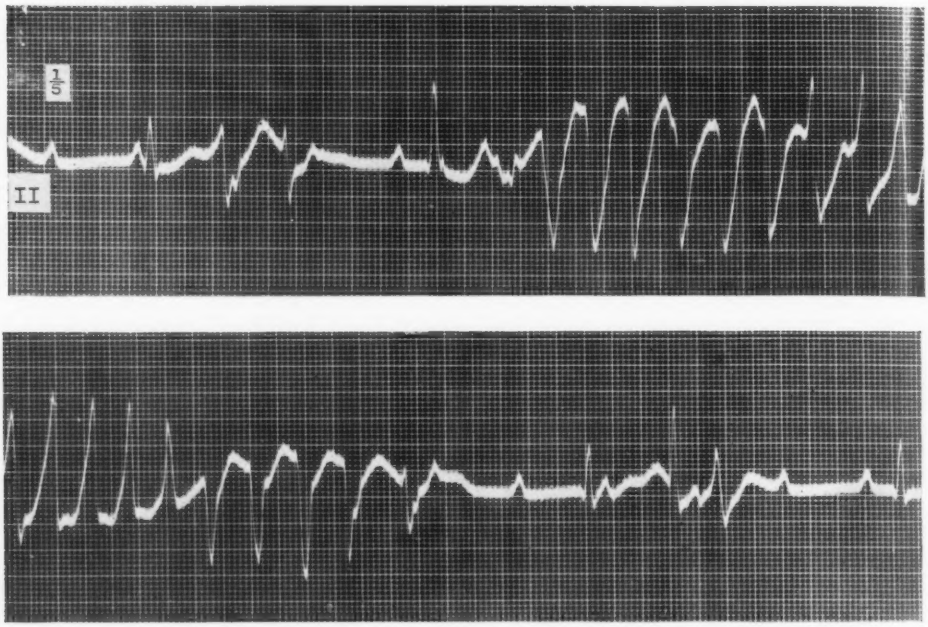

FIG. 11.—Case 8. Stokes-Adams attack. Paroxysm of high ventricular tachycardia (a continuous record has been divided). Complete heart block.

attack. Following the attack of ventricular tachycardia the electrocardiogram was much the same as that preceding it.

The above analysis applies to Fig. 11, taken while the patient was unconscious, and numerous other records were taken, some on continuous strips; they differed only in the duration of the high ventricular tachycardia and were never followed by ventricular standstill. Ventricular fibrillation appeared only in one record taken shortly before death.

DISCUSSION

Ventricular standstill

The attack was preceded in six of the reported cases by an acceleration of the auricular rate, but in many others and in our own case no sufficient record of the phase preceding the attack was available for reckoning. Hay (1921) called attention to " the gradual quickening of the rate of the auricle previous to

ventricular stoppage", and the matter is chiefly discussed by Wenckebach and Winterberg (1927), who found it in all three of their recorded cases, and by Condorelli (1932). As the auricular rate is so closely under nervous control, one might imagine that an increase of the auricular rate from exertion or emotion would often determine attacks. In Case 3 of Wenckebach and Winterberg, the emotion of an arithmetical exercise was observed to quicken the auricles and to cause ventricular standstill; but any such direct relation between exercise and emotion and the attacks must be rare. With the exception of that case, the rise in auricular rate in recorded cases has been spontaneous, and it originates in the auricle itself, probably in the sinus node. An increased auricular rate is known to worsen conduction to the ventricle, but its influence in the production of the standstill is unknown.

During ventricular standstill of short duration (20 seconds or less) auricular activity continues undisturbed or more often at a somewhat higher rate (Cases 2, 3, 4, 5, and 6). The presence of the P waves during ventricular standstill is an important distinction from a common faint, where with slowing there is a total cardiac standstill (Fig. 1). In the longer ventricular standstill of a Stokes-Adams attack the circulatory stasis affects the auricular activity. While the increase of the auricular rate during short ventricular standstill suggests an asphyxial irritation of the sinus node, the decrease of auricular rate after longer ventricular standstill (20 seconds or more) would suggest a subsequent depression in that node. Ectopic P waves may then appear often irregularly (Hermann, Froment, Gonin, & Mahaim, 1937; Géraudel, Laignel-Lavastine, & Boquien, 1933; Heimann, 1929), or P may disappear (Yater & Willius, 1929); alternatively auricular fibrillation and flutter may supervene (Wiltshire, 1923). Irregular and changing P waves have been observed, however, in short ventricular standstill (Cowan & Ritchie, 1935; Norris & Landis, 1938).

The cardiac standstill following a high ventricular tachycardia and fibrillation (Group II (*b*), Case 7) is often total. The effect of the high ventricular tachycardia (V.T.) and ventricular fibrillation (V.F.) on the sinus node is comparable with that of ventricular standstill, as the cardiac circulation is practically arrested. The occasional ectopic P waves seen in Fig. 9D (Case 7), is one effect of auricular stasis, and this and other effects of it are reported by Clerc and Lévy, Case 2 (1936), by Soulié (1938), and by Froment and Gonin (1938*a*), all with ventricular tachycardia and fibrillation (30 seconds or more) preceding the ventricular standstill. Sometimes the auricular rate during ventricular standstill (following high V.T. and V.F.) is accelerated (Lian & Deparis, 1934; Norris & Landis, 1938), and sometimes it is reduced (Gertz, Kaplan, Kaplan, & Weinstein, 1938).

Ventricular Tachycardia

A low ventricular tachycardia may *follow* ventricular standstill, but these cases are classed with Group I (Case 5). In our Case 6 (Group II (*a*)) it preceded ventricular standstill, increasing gradually from 55 to 75. The electrocardiograms between the attacks showed multiform and variable extrasystoles and varying bundle branch block (cf. Cohn & Lewis, 1913; and

Bain, 1941). Such aberrant complexes sometimes replaced the basic ventricular complexes at the low basic rate; at other times they composed a low ventricular tachycardia, with complexes like the extrasystole that followed the basic complex in fixed coupling. The same relation has been described by Herapath (1926) and others. This low ventricular tachycardia may be perceived by the patient as palpitation, as in Case 6, where it was the clear warning of the imminence of unconsciousness due to ventricular standstill.

Whether accompanied or not by ventricular standstill, a close affinity exists between the low and the high types of ventricular tachycardia. They often appear together or follow each other as in our Case 7, and in many of the published cases (see Tables II and III). We divided Group II into (*a*) and (*b*), because low ventricular tachycardia (up to 160) does not produce unconsciousness, but *precedes* a ventricular standstill that does; whereas high ventricular tachycardia with or without ventricular fibrillation does produce unconsciousness. The high ventricular tachycardia differs little from the low in the electrocardiogram, having regard to the rate which gives the undulations a less individual character. Any resemblance to auricular flutter is quite superficial, and there seems no fundamental reason for assuming a corresponding ventricular flutter with a distinctive mechanism as did De Boer (1923), and later Dressler (1929) and Scherf and Boyd (1940).

Ventricular Fibrillation

Confusion still exists between the high ventricular tachycardia and ventricular fibrillation. Yet, though either may follow the other—and the passage may be a gradual one—the electrocardiographic features are very different. The deflections of ventricular tachycardia are practically regular, and any variation in their shape or size occurs in phases. In any attack the highest rate (200–500) is found at the onset, and towards the end the rate may fall. The rate in ventricular fibrillation may be the same, higher, or even lower, but the deflections are always irregular in time, and constantly varying in shape and size, and so they resemble an artefact rather than a natural electrocardiogram (Fig. 9 A, B).

Schwartz (1936, *a*, *b*) divided the " transient recurrent ventricular fibrillation " (comprising the high ventricular tachycardia and ventricular fibrillation) into a pre-fibrillatory, fibrillatory, and post-fibrillatory period. The pre-fibrillatory period is one with multiple and variable extrasystoles and slower ventricular tachycardia (the auricles accelerating), and sometimes with a step-like increase of both auricular and ventricular rates with abrupt changes from partial to complete heart block. The fibrillatory period is that " resembling " ventricular tachycardia (i.e. the high ventricular tachycardia) and the phase of varying, irregular complexes (i.e. ventricular fibrillation). The post-fibrillatory period is the ventricular standstill that may last from 1 or 2 seconds (our Group III, here regarded merely as the expected pause after a paroxysm) to 20 or even 80 seconds (our Group II (*b*)), and the subsequent low ventricular tachycardia.

The instability and variability of the widened bundle branch block com-

plexes and of the associated extrasystoles pave the way for the installation of a new rhythm, which supersedes the A-V pacemaker and rules by its higher rate (low or high ventricular tachycardia or ventricular fibrillation). It is supposed that exhaustion of the new centres causes ventricular standstill during which they may recover some activity (post-fibrillatory ventricular tachycardia) until their final suppression by the basic rhythm.

The Stokes-Adams attack due to the high ventricular tachycardia (and ventricular fibrillation), and that due to ventricular standstill can only be recognized and distinguished by electrocardiogram, because the pulse and the heart sounds are absent in both varieties, and tachycardia may precede each (cf. Groups II and III). Although loss of consciousness of more than three minutes duration points to the high ventricular tachycardia (and fibrillation), our Case 3 with frequently recurring short attacks of ventricular standstill was barely conscious for hours. A late onset of unconsciousness, say that exceeding 20 seconds after the disappearance of the pulse and the heart sounds, is the rule in ventricular tachycardia (and fibrillation) according to Schwartz (1936*a*). But unconsciousness in ventricular standstill may certainly be delayed for over 10 seconds (Cases 1 and 4); and short attacks of ventricular tachycardia (6 seconds in Case 8), if repeated, may induce long periods of unconsciousness. Again, though circulatory arrest is the most important determining factor in the duration of unconsciousness, other factors may contribute, e.g., the rapidity of onset of block and the fall in rate, the frequency of the attacks, a low blood pressure (as after cardiac infarction), and the state of the cerebral arteries. Such being the situation, it is difficult to state the duration of circulatory arrest that produces syncope or convulsions, especially now we know that Stokes-Adams attacks are not due simply to ventricular standstill, and that tachycardia as well as standstill may be in operation. Though admittedly approximate, the onset of unconsciousness in ventricular standstill is given by Mackenzie (1925) as after 10 seconds, and of convulsions as after 15 seconds; by Lewis (1925) as 3–5 and 15–20 seconds; and by Froment and Gonin (1938*b*) as 5–15 and 30–45 seconds respectively.

The electrocardiogram of the Stokes-Adams attack under special conditions.

The grade of heart block between the attacks, whether *partial* or *complete*, has no bearing upon the electrocardiographic features of the attack. Our Groups I, II, and III contain an almost equal number of both grades.

Among 10 cases of *paroxysmal heart block*, the attack was due to ventricular standstill alone (Group I) in 8 (Yates and Willius, 1929; Cheer and T'ang, 1932; Sachs and Traynor, 1934; Schwartz, 1936*c*; Hermann, Froment, Gonin, and Mahaim, 1937; Sigler, 1939*a*; Teran, 1941; our Case 4, Fig. 6). In one the ventricular standstill was preceded by the low ventricular tachycardia (Group II (*a*)) (Gager and Pardee, 1925), and in the remaining case it was preceded by the high ventricular tachycardia (Group II (*b*)) (Gertz, Kaplan, Kaplan, and Weinstein, 1938). Thus ventricular standstill seems to be the usual basis of the Stokes-Adams attack in paroxysmal block. Comeau (1937) has discussed paroxysmal heart block as a whole.

No actual electrocardiogram of a Stokes-Adams attack in *congenital heart block* has been found, though Fæssler (1939) has collected 8 such reported cases, and 6 had established complete heart block between the attacks. In his own case there was no block, but nodal rhythm during the unconsciousness.

The incidence of heart block in *coronary thrombosis* is given as 7·6 per cent Kerr (1937), and as 3·2 per cent by Master, Dack, and Jaffe (1938). One of Kerr's 12 cases had Stokes-Adams attacks; and 3 of the 5 cases of Master, Dack, and Jaffe were comatose or semi-comatose, though without distinctive Stokes-Adams attacks. Among 45 cases with Stokes-Adams attacks, 15 were due to posterior coronary thrombosis according to Schwartz (1936*b*). Among the cases we have collected, recent coronary thrombosis was present in 6. In 3 (Laufer, 1934; Schwartz, 1936*b*; Scherf & Boyd, 1940) the Stokes-Adams attack was due to ventricular standstill (Group I), in 2 (Freundlich, 1932; Soulié, 1938) it was due to ventricular tachycardia and fibrillation with standstill (Group II (*b*)), and in 1 (Sigler, 1939*b*) to ventricular tachycardia and fibrillation (Group III). Except in Soulié's case (with variable bundle branch block), the electrocardiogram was of the T_3 type. Although no statistical conclusions can be drawn from 6 cases, it is evident that Stokes-Adams attacks in coronary thrombosis may be due to ventricular standstill, to ventricular tachycardia and fibrillation, or to these combined, and that all are not due to ventricular fibrillation.

Prognosis

The nature of the electrocardiogram during attacks has a great influence upon the prognosis, and this is evident from the 35 cases that were followed up; 15 belong to Group I (ventricular standstill alone) and 20 to Groups II and III (low or high ventricular tachycardia and fibrillation, followed or not by ventricular standstill). Among the 15 of Group I, 5 died during the observed attack and 7 were alive and attack free over a period of from three months to four and a half years; in 3 death was due to other than heart conditions (cerebral thrombosis, peritonitis, and spinal tumour). Among the 20 of Groups II and III, 16 died in an attack (11 during the observed attack and 5 within a year of discharge from hospital), and only 4 were alive at the end of three to ten months. It follows that patients showing ventricular standstill alone have a fair chance of recovery even after severe Stokes-Adams attacks, but that when the attacks include the low or high ventricular tachycardia (and fibrillation) the immediate prospect of recovery is far worse, and even if they recover they seldom survive for more than a year.

Summary

Stokes-Adams disease is a name applicable to patients with heart block who suffer from recurrent attacks of loss of consciousness due to ventricular standstill, ventricular tachycardia, ventricular fibrillation, or a combination of these.

During a Stokes-Adams attack from ventricular standstill the auricle con-

tinues to beat, whereas in cardiac syncope of other types there is as a rule total cardiac standstill.

Cardiac syncope of neurogenic origin (e.g. ordinary fainting, and ventricular standstill from disease affecting the vagus or carotid sinus) and cardiac syncope of myocardial origin without heart block (e.g. in nodal bradycardia and in paroxysmal ventricular tachycardia) are excluded by this definition, though there are borderline cases.

(2) The cardiac mechanism of Stokes-Adams attacks was studied on electrocardiograms recorded during the period of unconsciousness, in 8 of our own cases and in 56 reported cases. These fall into four groups or types and are tabulated according to the electrocardiographic basis of the attack.

Group I (28 reported cases and 5 of our own) includes those with *ventricular standstill alone* (Table I).

Group II (16 reported cases and 2 of our own) includes both those with (a) *low ventricular tachycardia* and (b) *high ventricular tachycardia and fibrillation, when either is followed by ventricular standstill* (Table II).

Group III (12 reported cases and 1 of our own) includes those with the high *ventricular tachycardia and fibrillation* without ventricular standstill (Table III).

Group IV includes those rare cases with *extreme bradycardia in heart block* (no Table).

It is evident that ventricular standstill alone is not the only cardiac lapse that determines a Stokes-Adams attack. It is often due to ill action, not to inaction of the ventricle. Ventricular standstill is responsible for about 55 per cent ; ventricular tachycardia (with or without ventricular fibrillation), followed by ventricular standstill, for 25 per cent; and ventricular tachycardia without ventricular standstill for 20 per cent.

(3) *Ventricular standstill* is sometimes consecutive to a rise in the auricular rate. As exertion or emotion so seldom determines ventricular standstill, this increase in the auricular rate probably originates locally in the auricle and not from any nervous influence.

During a short ventricular standstill (below 20 seconds), the auricle beats regularly, often at an increasing rate, and the persistence of P waves during the ventricular standstill is a feature distinguishing it from the total standstill of cardiac syncope. During a long ventricular standstill (above 20 seconds), or when ventricular standstill is preceded by high ventricular tachycardia and fibrillation (Group II), the auricle may show slower, irregular, and ectopic P waves, auricular fibrillation and flutter, or it may even stop.

(4) In the group with *ventricular tachycardia*, multiple and variable extrasystoles and varying bundle branch block complexes between the attacks are common, as might be expected. Low ventricular tachycardia (up to 160) does not produce unconsciousness, but it provokes the subsequent ventricular standstill that does produce it (Group II (*a*)). High ventricular tachycardia and fibrillation (200–500) produce unconsciousness (Group III), and this may be prolonged by the subsequent ventricular standstill (Group II (*b*)). The electrocardiogram of ventricular tachycardia is composed of regular deflections like bundle branch block, which at high rates merge into simple undulations;

yet the term " ventricular flutter " need not be used, for the resemblance to auricular flutter is superficial.

(5) *Ventricular fibrillation* is distinguished from ventricular tachycardia by its irregularity both in rate and in form, though the rate per minute may be the same, higher, or even lower. High ventricular tachycardia easily passes into fibrillation, which ends with ventricular standstill or with gradual resumption of the basic rhythm through a period of low ventricular tachycardia or of varying extrasystolic complexes.

(6) The essential basis of an attack can only be decided by electrocardiogram. The prolongation of unconsciousness or its late onset in ventricular tachycardia and fibrillation cannot distinguish this group from that of ventricular standstill alone, because other factors may influence unconsciousness, e.g., the suddenness of the development of block or of the fall in rate, a rapid succession of attacks, and the state of the cerebral arteries. For similar reasons it is difficult to state exactly what must be the duration of the circulatory arrest to produce syncope or convulsions.

(7) In established complete heart block or in partial heart block the Stokes-Adams attack may belong to any group, whereas in paroxysmal heart block it is generally in Group I, i.e., ventricular standstill alone. After coronary thrombosis, attacks due to ventricular standstill alone may occur, as well as those due to ventricular tachycardia and fibrillation.

(8) No prognostic significance can be attached to the grade of heart block, partial or complete, obtaining between the attacks, but the electrocardiographic nature of the attack has great significance in prognosis—and doubtless in treatment, though this is not here considered. Patients with ventricular standstill (Group I) have a fair chance of recovery and often survive for many years, whereas those with ventricular tachycardia and fibrillation (Groups II and III) seldom recover and then rarely survive for more than a year.

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TOTAL THYROIDECTOMY FOR HEART FAILURE: AN UNUSUAL CASE

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Congestive cardiac failure associated with thyrotoxicosis is almost always relieved by sub-total thyroidectomy. It is probable that the improvement in the cardiac condition can be attributed to two distinct factors: first, to the removal of a direct toxic action of the thyroid secretion on the heart, and, secondly, to the removal of the burden of an augmented circulation rate that the increased metabolism of thyrotoxicosis places upon the heart. The direct effect of the thyroid on the heart is evident in the common association of auricular fibrillation with thyrotoxicosis and in the frequency with which normal rhythm is restored in such cases after sub-total thyroidectomy. The close connection between thyrotoxicosis and auricular fibrillation is also seen in the common occurrence of the latter in the first two days after thyroidectomy —a time when all the manifestations of thyrotoxicosis are generally at their height.

The effect of thyroidectomy in sparing the work of the heart has been most fully demonstrated by the work of Blumgart, Levine, Berlin, and their colleagues on total thyroidectomy for non-thyrotoxic heart disease. In a series of papers (summarized by Blumgart *et al.* 1933) they showed that in the absence of cardiac failure the circulation rate in normal subjects and in cases of thyrotoxicosis and of myxœdema was adjusted in proportion to the metabolic rate. When heart failure is present, however, this relationship breaks down, so that in the ordinary cases of congestive heart failure, although the basal metabolic rate is normal, the circulation rate is no greater than in a case of myxœdema without heart failure. Similarly, in cases of thyrotoxicosis with heart failure, the circulation rate is less than in other such cases without heart failure. On the basis of these findings, they put forward the practical suggestion that in congestive cardiac failure the lost equilibrium between circulation rate and metabolic rate could be restored by reducing the latter to the level of the former by total thyroidectomy. Although the measure of success achieved by this therapy is still in dispute, it seems clear from the work of Blumgart and his colleagues, and those who have repeated it, that cases of congestive cardiac failure can sometimes be improved by total thyroidectomy; and that such improvement,

when it occurs, is brought about by diminishing the work the heart has to do rather than by increasing its capacity to do it.

We are reporting here the case of a patient in whom the removal of a normal thyroid gland was followed not only by the relief of cardiac failure but also by the disappearance of auricular fibrillation. It seems to us improbable that the abolition of auricular fibrillation can be attributed merely to the lowering of the metabolic rate, and we feel bound to postulate in this patient a direct action of the normal thyroid gland on the heart. However it was brought about, there can be no doubt that total thyroidectomy proved to be a most successful form of therapy, and one which might be worth trying in similar cases.

The patient was a man, aged 51, at the time of operation. He was first admitted to University College Hospital under Dr. Kenneth Harris in 1931, at the age of 43, with a history of breathlessness and swelling of the legs for four weeks. There was no history of rheumatic fever. On admission, he was found to be in congestive cardiac failure; the heart rate was 160–180 and the rhythm regular. A cardiogram showed auricular flutter. He was given digitalis in full doses; the rhythm became normal on the seventh day and the signs of congestive failure then quickly disappeared. He left hospital with a heart of normal size (orthodiagram) and a regular heart rate of 70–86.

He then continued at full work (clerical), with occasional attacks of palpitation as the only symptom, until Christmas 1938, when severe palpitation developed, leading to his readmission to hospital four weeks later. He then showed signs of early congestive cardiac failure; the heart rate was 140–170 and the rhythm grossly irregular. A cardiogram confirmed the diagnosis of auricular fibrillation. There were no signs of valvular damage. He was treated with digitalis; the heart rate was reduced to 84–96 and the signs of congestive failure disappeared; but auricular fibrillation persisted.

He was, however, unable to return to work, and when seen in March 1939 fibrillation was still present and the heart rate was 148. In May 1939, he was readmitted in congestive cardiac failure. The heart rate was 100–140 and auricular fibrillation was still present (confirmed by cardiogram). The heart was considerably enlarged. He was given digoxin intravenously, and thereafter by mouth. In three weeks the signs of congestive failure had disappeared and the heart rate was 80–96. He was specifically examined at this time for signs of thyrotoxicosis; but none were found.

At this point Dr. Harris advised a total thyroidectomy, and on June 1, 1939, this was performed under local anæsthesia by Mr. Julian Taylor. The post-operative course was uneventful, except for the occurrence of attacks of tetany on the eighth, tenth, and thirteenth days; these were easily controlled by injections of calcium gluconate and by calciferol by mouth. On the thirteenth day after operation the heart returned to normal rhythm (confirmed by cardiogram): this persisted for the rest of his stay in hospital, the rate being 68–76. The heart returned to normal size. He was given no thyroid extract, but calciferol was continued to control the tetany.

When next seen, at the end of July, he was back at full work, and the rhythm was still regular. In September, he was still at work, though somewhat easily tired. The heart rate was 82, and regular. So far he had had no thyroid extract, and by this time the signs of myxœdema were obvious and appeared to be troubling him. Tab. thyroid sicc. (B.P.) was therefore started in doses of 1·0 gr. daily.

In October the heart was still regular and the rate 84. The dose of thyroid was now increased to 1·5 gr. daily and maintained at this level till January 8, 1940. In November 1939 he had an attack of tachycardia with irregular rhythm which lasted several hours. In December there were several similar attacks. On January 8 he was seen in an attack of tachycardia; the rhythm being regular and the rate 160. It was presumed

that this was paroxysmal auricular flutter, but a cardiogram could not be taken at that time. The thyroid extract was then stopped. A week later he was seen again. This time auricular fibrillation was present, the heart rate being 110. Two days later the heart became regular again and tachycardia ceased. One further attack with irregular heart action occurred on January 21; but it proved to be the last.

By March he had been without thyroid extract for ten weeks and the symptoms of myxœdema had become troublesome again. He was therefore restarted on 0·5 gr. daily. This proved to be enough to keep the symptoms of myxœdema at bay. He has been seen at regular intervals for a year since restarting thyroid extract and there has never been any suggestion of a recurrence of the paroxysmal flutter or fibrillation. He has been at full work during this period, and recently has been fire-watching as well. He was anxious to join the Pioneer Corps, but was advised not to do so. He is able to take a normal amount of exercise without becoming breathless.

The thyroid gland from this patient weighed 20 g., which is within normal limits for this country. It was normal in appearance, both macroscopically and microscopically. The Golgi apparatus in the cells lining the vesicles was not enlarged.

Discussion

It will be seen that this case differs from those recorded by Blumgart and his colleagues in that total thyroidectomy not only relieved congestive cardiac failure but also restored the heart to normal rhythm. In fact, the removal of a normal thyroid in this case had the same effect as the removal of an abnormal thyroid in a case of thyrotoxicosis. This similarity is the more striking in that in our case normal rhythm returned on the thirteenth day after operation; it is at about this time that auricular fibrillation commonly disappears after thyroidectomy in thyrotoxic cases. Moreover, in our case, as in many cases of thyrotoxic heart disease, cardiac failure only appeared when uncontrolled auricular flutter or fibrillation was present; and it disappeared promptly whenever the heart rate was brought under control. Cardiac failure was dependent on the presence of the disorder of rhythm. In Blumgart's cases, on the other hand, auricular fibrillation was not abolished by total thyroidectomy, and cardiac failure was relieved in spite of the persistence of the disorder of rhythm.

These facts made us consider the possibility that our case was in fact one of thyrotoxicosis. But in our opinion the finding of a thyroid of normal size and normal histological structure excludes this.

If we accept this conclusion, we are faced with the surprising conclusion that thyroidectomy in a man with normal thyroid function has had the same effect on the heart as thyroidectomy in a case of thyrotoxicosis. Our suggested explanation is based on the widely held opinion (Lahey, 1929; Hurxthal, 1931; Lewis, 1931; Andrus, 1932; Rosenblum and Levine, 1933; Mayer and Sittler, 1936; Means, 1937; and Gotta, 1938) that thyrotoxic heart failure is due to a combination of two factors—a primary cardiac disorder and thyroid toxæmia. On this view, thyrotoxicosis cannot by itself cause cardiac failure: it can only make manifest a cardiac disorder that would otherwise remain latent. If we accept the existence of such latent cardiac disorders, it is not difficult to imagine that similar conditions of the heart might exist that are manifest at a normal level of thyroid function but become latent at a low level. This interpretation is supported by our experience of a case of toxic nodular goitre, in which sub-

total thyroidectomy was followed by the disappearance of auricular fibrillation and later by the development of myxœdema; doses of thyroid extract sufficient to relieve the symptoms of myxœdema led to the reappearance of auricular fibrillation. Swan (1924) had a similar experience. It will be recalled that the patient described here behaved in a similar way, developing paroxysmal auricular fibrillation on 1·5 gr. of thyroid extract a day, but not on 0·5 gr. a day. The behaviour of these cases suggests that, in a suitably predisposed heart, auricular fibrillation may be precipitated not only by excessive amounts or by an abnormal type of thyroid hormone but also by the normal hormone in normal quantities.

If this interpretation is correct, then the success of thyroidectomy in our case was due not, as in Blumgart's cases, to a reduction in the work which the heart had to do but to the removal of an agent injurious to the heart. As thyroid extract in full dosage caused the reappearance of auricular fibrillation and omission of the extract was followed by a return to normal rhythm, this agent is presumably the thyroid secretion itself. These considerations make it important to define as far as possible the type of case in which a similar result is likely to be obtained. Among the cases with auricular fibrillation described by Blumgart *et al.* (1935) there is no record of any in which normal rhythm was restored after total thyroidectomy. All these cases had long histories of cardiac disease and all showed evidence of structural damage of the heart. It seems likely, therefore, that the abolition of auricular fibrillation will not be achieved when it has been long in existence or where it is accompanied by structural damage of the heart. This conclusion is borne out by the experience of Rosenblum and Levine (1933) in cases of thyrotoxicosis with heart failure. They found that of 11 cases with auricular fibrillation and mitral stenosis, only 1 returned to normal rhythm after sub-total thyroidectomy; whereas of 6 cases with auricular fibrillation without evidence of structural damage to the heart, all 6 returned to normal rhythm. Furthermore, the only report of cases similar to ours that we have been able to discover is that of Singer (1937); in both of his cases paroxysmal auricular fibrillation disappeared after total thyroidectomy. Therefore, it seems that the type of case likely to benefit from total thyroidectomy in the way ours has done is one in which auricular fibrillation or flutter is either paroxysmal or not of long duration, and there is no evidence of structural damage of the heart. Such cases are usually regarded as suitable for quinidine therapy; but it may be found that in some instances total thyroidectomy is a more stable and satisfactory alternative. The mortality from the operation is likely to be small, for Berlin's (1935) last 62 total thyroidectomies were performed without fatality, although his cases had gone through long periods of congestive cardiac failure and were therefore poorer operative risks than the type of case we have in mind.

Summary

A case has been described in which the complete removal of a normal thyroid gland was followed by the disappearance of auricular fibrillation and

congestive cardiac failure. Auricular fibrillation reappeared when the patient was given 1·5 gr. of thyroid extract daily, but disappeared again when the dose was reduced to 0·5 gr. daily.

It is suggested that in this case the normal thyroid hormone had the same effect on the heart as has the excessive or abnormal secretion in cases of thyrotoxicosis.

It is suggested that cases of auricular fibrillation or flutter, in which the disorder of rhythm is either paroxysmal or of short duration and there is no evidence of structural damage to the heart, might be expected to benefit in a similar way as the result of total thyroidectomy.

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